ABSTRACT

OF

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Annual Meeting of the Association
of Life Insurance Medical
Directors of America

VOL. XXVII

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An Abstract of the Proceedings of the Association of Life Insurance Medical Directors of America

FIFTY-FIRST ANNUAL MEETING

The Fifty-first Annual Meeting of the Association of Life Insurance Medical Directors of America was held at the Hotel Statler, Boston, Massachusetts, on October 17 and 18, 1940.

The following members, delegates and guests were present at some time during the sessions: Drs. J. W. Abbott, W. J. Allison, H. H. Amiral, H. H. Amsden, T. D. Archibald, E. M. Armstrong, W. B. Aten, G. W. Bachman, H. A. Bancel, N. J. Barker, W. B. Bartlett, R. A. Behrman, M. B. Bender, F. P. Bicknell, E. B. Bigelow, J. R. Biggs, W. F. Blackford, D. N. Blakely, J. E. Boland, William Bolt, W. M. Bradshaw, J. L. Brooks, C. T. Brown, F. R. Brown, H. B. Brown, B. F. Byrd, J. H. Campbell, L. D. Chapin, C. L. Christiernin, E. A. Colton, D. B. Cragin, H. W. Crawford, H. C. Cruikshank, R. M. Daley, W. L. Davis, A. H. Davison, R. P. Dawson, E. J. Dewees, E. G. Dewis, T. H. Dickson, H. W. Dingman, J. P. Donelan, J. G. Downing, O. M. Eakins, W. R. P. Emerson, J. L. Evans, W. G. Exton, J. G. Falconer, H. H. Fellows, W. E. Ferguson, R. M. Filson, H. M. Frost, D. S. Garner, E. E. Getman. J. M. Gilchrist, R. T. Gilchrist, R. A. Goodell, R. J. Graves, George Greenway, F. L. Grosvenor, Llewellyn Hall, Frank Harnden, L. E. Hathaway, J. K. P. Hawks, W. D. Heaton, O. C. Hendrix, C. E. Herron, F. R. Holbrook, C. O. Hollinger, Byam Hollings, J. C. Horan, J. L. Humphreys, J. R. B. Hutch-

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Total attendance at all sessions, 166.

PRESIDENT FROST—We are indeed fortunate to have Dr. Frank H. Lahey talk to us this morning. He needs no introduction to anybody. He has appeared on our program before, and is well known to us. I know him somewhat personally. I appreciate his friendship and I have a very high admiration for him.

As you know, he is President-Elect of the American Medical Association. He has very graciously consented to come here this morning, and I know he is a busy man. He is leaving shortly for Cleveland, Chicago and points west, to be gone several days. But, he did consent to come and bring you his greetings and the greetings of the American Medical Association. Dr. Lahey!

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Dr. Frank H. Lahey—Mr. President and Members of the Association. I feel a little strange in this role; I am not quite used to it yet. I recently spoke for the first time in the capacity of President-Elect of the American Medical Association, before the Middlesex South District Society. I have felt quite at home, always, in discussing medical subjects, and I expect that within a reasonable time, I will feel quite at home on this mission. But, I am frank to admit to you that I do not possess quite the confidence and the assurance in discussing some of these problems that I have in discussing medical problems. On the other hand, I have grown up, I believe, in the proper way, at least, to know something about this subject, just as I believe I have grown up in the proper way to know something about the subject of medicine. I mean that it is most difficult not to be politically contentious today, and compare one's up-bringing with the political candidates, and so on; but, at any rate, if I can avoid political reference, I will say that I have really grown up the hard way, which is the best way.

If I were to make a criticism of our method of life, today, I would certainly direct it along that line.

And so, I have grown up the same way with the American Medical Association. I mean that I have grown up through the Secretary-ship of the Surgical Section for four years, developing the program, by correspondence with everybody interested in presenting papers throughout the country, to the Chairmanship, to Membership on the Council on Scientific Assemblies, to Membership on the Council on Medical Education and Hospitals, and finally to this position, unsought by me now.

That, I present, not because I wish to elevate myself at all, but because I wish to bring a point up in connection with it. It seems to me that one of the things we do in this generation is to be unappreciative of what we have.

It is so easy to cast one's eyes longingly on the distant horizon, where I have repeatedly said images are still obscure and perhaps pleasing to the eye, in prospect of the future, but it is not clear as to some of the disadvantages and drawbacks. That is why I say that we must be very careful, just as we are critical of many things about our country, to be appreciative of what we have in the American Medical Association. We must, likewise, I think, be very very careful that we, as members, and particularly those of us who are officers of the American Medical Association, do not become so obsessed with its and our greatness that we are blind to some of its defects, and that we are sensitive to its criticism.

Now, I think one of the things that I should do, and one of the messages I should bring to you, is that I, personally, think that one of the great things that can happen to me as an individual, or to an Association such as the American Medical Association, is to get plenty of criticism. That is the most helpful state of being that can exist, I think, regarding such a large organization.

One of the things we have to be extremely careful about, I think, is that its officers and its representatives do not become bureaucratic.

Therefore, I say that these things which have come about within recent years—that is, these criticisms—are excellent

things, and they are the test of the broadness of the American Medical Association, its officers and its representatives, as to how well they accept and with what little irritation they listen to criticisms of the American Medical Association.

I have repeatedly said about myself, that having grown up in this school of hard knocks, or practical life, one of the very first things I have had to learn—and most of the things I have learned have come from being really and actually hit over the head—is that when I am criticized, certainly the first thing I ought to do, if I wish to be fair to myself and my critic, is to sit down and analyze the criticism with the conviction that there may be possibly some justice to it.

At any rate, you will probably remember what George Vincent said. He said: "A great many people don't reason; they just re-arrange their prejudices."

Now, if I react to criticism in that way, I am not doing myself or my critic justice.

There are one or two points regarding this question of the American Medical Association that I would urge. One of them is that if any one feels critical, he should at least do himself and the Association the justice of investigating what it does, and investigating its conduct.

After all, it represents the most democratic organization that can be possibly developed for you and for me. It represents reputation; it represents conduct of affairs by representatives who are elected by the various state societies. And, if things go wrong, it is really largely our fault, as individuals, if we do not do something in the way of urging our representatives to air our viewpoints and correct them.

One of the things I promise is that other than these statements of fact, it will never be my purpose, as President-Elect or President of the American Medical Association, to be responsible for contentious orations. I am sure that that is a very bad thing.

I believe that the American Medical Association has accomplished enough. I am sure you are familiar with the figures. Think of the diphtheria mortality, which you know so well,

of 1.5 in this country as compared with 9.6 in other countries. Think of the relation with the doctor, with which you are familiar, one doctor to every 600 people, as compared with one to every 1,300 in Germany, one in 1,000 odd in England, and one in 3,500 in Czecho-Slovakia.

These are just a few of the things that medicine has accomplished.

Think of the number of available beds that we have in this country, 1,119,000. And remember, also, when we are critical and we think we need new hospitals, that many of these are vacant all the time and should be utilized first.

These are the facts which I think should always be presented, and not contentious and bitter and acrimonious replies to criticism. All that does is build up strife, and that gets one nowhere.

Now, as to what medicine has done, we all know the value of the American Medical Association. But, let us take one of the most outstanding things. Where would we be today in preparedness without the aid of the compiled information and the information which is daily being compiled regarding the availability of doctors? You all know that the American Medical Association, through its Committee on Preparedness, of which I automatically become a member and have to go to all the meetings and know its progress, sent out 179,000 questionnaires. You know that it is in the process of so organizing these replies in a punch-card system that it will be possible to supply the government immediately with information which will later on undoubtedly be requested as to the types of orthopedists, general surgeons, and so on, who will be available.

That is but a small example of what the American Medical Association is doing and how valuable it is.

As a member of the Council on Medical Education, I am very much aware of what the effect of the American Medical Association has been upon medical education. I realize that the number of medical schools in this country has been cut

down two-thirds in number within the last few years, and that it has accomplished the elimination of the second and third-rate medical schools, and that in the face of this cut-down, there are now seventy-seven as compared with a little over one hundred, and there are many more and many better doctors turned out.

These things are not accomplished without every effort. Remember that the American Medical Association maintains and pays for a staff of investigators, who travel the country, investigating medical schools, investigating hospitals, reporting on their soundness. All this goes on, I think, almost without our being conscious of it.

I have said to the American Medical Association that I thought one of the defects is that they do not get credit enough for the work that is done, because they do not publicize it in a way that attracts the eye and the interest of the general medical profession. But, the American Medical Association is really doing a great thing.

Now, the one thing that I would urge strongly before I close is that when we have men who do feel critical of the American Medical Association, they should pay a visit to the House of Delegates to see how long and how well the members of the House of Delegates and these sub-committees work.

I have had to go to everything, to all the meetings of the American Medical Association. The delegates are usually there on time. I usually get there Sunday night and stay there until Friday night, as a rule, and I have known what these men do. The men on these various committees, such as the Committee on Education, the Committee on Public Relations, the Committee that has to do with Ethics, very often do not even get a chance to go and look at the exhibits; they don't hear the papers that are read; at least many of them don't. They get up in the morning, and sit in the House of Delegates and they really work, and if there are critics of the American Medical Association, the best way to really work

out whether or not their criticism is sound and just is to go and see how these men work and ask for the opportunity to present their criticisms before this group of men who, I know, will welcome them, and, if they are reasonable, will do their very best to adjust them.

Of course, I, as President-Elect, am only an individual trying to learn the ropes; I mean that I get a year of apprenticeship in this job in order that when I come, as your representative, to be President of the American Medical Association, I will not be quite as much of a tyro as I am bound to be in some measure.

And so I do bring the greetings and the best wishes of the American Medical Association, and I wish you every success in this meeting, which I know you will have.

PRESIDENT FROST—We are certainly thankful for Dr. Lahey's coming here this morning, and we, in turn, pass our greetings to the American Medical Association.

I have another distinct pleasure; I know it will be a pleasure for you and it is certainly a pleasure for me. I am blessed by having a Chief who is also a friend. We have much in common. We have tramped the mountains together; we have fished together; we know each other pretty well, and we work together.

Without further introduction, I present to you for a word of welcome, Mr. George Willard Smith, the President of my company!

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MR. GEORGE WILLARD SMITH—In accepting the invitation of your President and my friend, I thought I would get that in first, I do it with a peculiar pleasure, because I understand that this is the first time that the Association of Medical Directors has ever met in Boston, and I do believe it is high

time you came to old Boston, in your Fifty-first year!

Boston has a reason for your presence, and we are proud of the medical traditions which are associated with Boston, with its Harvard Medical School, founded almost one hundred and sixty years ago, in 1762, with the Massachusetts General Hospital, which has had a long sphere of influence since 1811, and I presume that Boston perhaps is best known in medical circles because of the first demonstration of anesthesia by ether, which I believe came in 1846, about the time that my company was started.

We started in 1843. And at this point, just a word about the company. At the time we started, there was no such thing as mutual life insurance, and our great and General Court, called the Massachusetts Legislature, thought that to be on the safe side, it would be advisable to provide in our charter a provision that one-third of the profits of this new venture of life insurance should revert to the Massachusetts General Hospital. That is a part of our original charter. As a matter of fact, we did pay a very small amount—I think it was $166.67—for a period of three years, to the Massachusetts General Hospital during the first three years of our existence.

So, we do have something in common with the medical history of Boston, aside from our great interest in your organization and our appreciation of what you have done for life insurance.

You are specialists in scientific medical selection, and you have brought the science of medical selection to life insurance in a very practical manner.

I do want to emphasize a point this morning that perhaps never in the history of your Association, and certainly in the history of life insurance, has scientific medical selection been more important. I say this because of certain other factors which enter the life insurance picture.

Certainly, there are factors which are beyond our control, and the chief one is the steady decrease of the yield on investments, a factor which is beyond the control of Manage-

ment, and which is beyond the control of any one of the companies, but which affects all of the companies.

As the decrease in earnings comes largely through the decrease in the yields on our investments, we all look with greater interest and with a profound sense of satisfaction at the contributions to surplus from mortality as they come to us through the scientific selection, not alone of medical directors, but of the medical directors associated with our actuaries and our underwriters and with all management policy.

And so I do believe that, in this meeting which you have come together for, you will take one step ahead again, in scientific selection, and in meeting the problems that currently arise in medical selection.

I believe in life insurance, and I know you believe in life insurance, and I believe that 64,000,000 policyholders have a profound faith in the institution which you have helped to create and to make safe. I speak of this 64,000,000 group as the thrifty half or the prudent half of our population, because they have the real spirit of American independence. They are not satisfied to rely upon government promises for their future independence or for the dependence of their beneficiaries. They have decided through self-denial and through foresight to provide for their own independence. And that brings up the idea of how they can do it safely, and through your efforts. Through your efforts, we have a vehicle which has proved sound through all the tests of time of the last one hundred years, a vehicle which will provide independence for millions of Americans.

It has become an American custom in which we all take pride. I wish to say again that I am delighted to have you here in our crisp, autumn weather. It may have been cold last night, but I am delighted to have you here at this season of the year, when you can see some of the glories of the foliage in this part of the country, and to wish you every success in your meeting.

I thank you.

PRESIDENT FROST—Dr. Christiernin has a matter he wishes to bring before this group at this time.

DR. CHRISTIERNIN—Last summer, our President assigned me to detached duty in Washington. The Surgeon-General, Dr. Parran, proposed to organize a committee, and he felt the insurance business should be represented, and Dr. Frost asked me to take over that assignment. I have a little, brief, statement that was sent out early in the procedure, which will tell you about what the problem is.

The National Institute of Health of the United States Public Health Service is organizing a new unit for research into some of the many problems of aging. With the conspicuous shift to greater age in the population, senescent individuals are becoming increasingly significant in the national economy and defense. Preventive medicine must attack the practical problems of the rising proportion of deaths attributable to diseases of middle and later life and energetically attempt to augment the health and vigor of those past the meridian. Aging is a continuous biologic phenomenon which starts upon the creation of a new individual and continues at variable rates until death. The problems of aging (gerontology) are not limited to the diseases of the aged (geriatrics), for the latter are the consequences of senescence. In man probably the most significant period of life for gerontologic study is late maturity, approximately the two decades between 40 and 60.

The problems of aging are logically divisible into three major fields of investigation: (1) the biology of senescence as a process, (2) the human clinical problems of aging and of diseases characteristically associated with advancing years which include the mental changes of senescence and senectitude as well as the physical changes, and (3) the socio-economic problems of a shifting age distribution in the population. The National Institute of Health is concerned with the first two of these divisions of the science.

What I wish to say here is that some of the companies may have been conducting investigations or studies over the years,

which may be of service and help to this Committee in its studies.

By the way, this Committee is called the Committee on Gerontology, and I didn't know what it was until I looked it up.

I remember years ago, we tried to find out something about the relation of stem length to mortality. Much to our surprise, we found that Dr. Muhlberg's company, the Union Central, had been doing that work for years, and had over 100,000 records. So that there may be among us others who have been doing special work, and if you have anything of that sort in your files or records that you think might be of help, I wish you would let me know so that I can pass it on to this Committee.

I thank you!

PRESIDENT FROST—I had a very pleasant experience this spring. I made a hurried trip to Colorado Springs, as your official at the American Life Convention. I had visited that convention once previously some fourteen years before, as I recall it. I have always admired the character of their program. I had heard that they believed in interspersing a little play with the scientific matters. While my stay was short, I thoroughly enjoyed the spirit of that younger association. It gives me a particular pleasure to welcome the Chairman of the Medical Section of the American Life Convention, Dr. Robinson, who brings you the greetings from his organization.

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DR. A. J. ROBINSON—Mr. President and Members of the Medical Directors' Association. It is particularly pleasant for me to bring you greetings from the Medical Section of the American Life Convention, and to extend to you a cordial invitation to attend our next meeting. Some preliminary work has been done on the program, which is in the very capable hands of Dr. John M. Livingston, who is Director of the

Mutual Life of Waterloo, Ontario. I promise you an interesting and instructive scientific program. Those of you who have attended previously our Medical Section meetings know that they are arranged so that our scientific sessions are held in the morning, and afternoons are devoted to becoming better acquainted with our associates in this business.

The meeting is to be held at the Homestead Hotel, Hot Springs, June next, on the 17th, 18th and 19th. Medical Directors and their associates of companies who are not now members of the Medical Section are cordially invited, and I hope to see many of you at our meeting next June.

Thank you very much for your attention to these few words!

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PRESIDENT'S ADDRESS

It is with the keenest of pleasure that I welcome you to the City of Boston; not only in my own behalf, but also in behalf of all the members of the medical staffs of the four life companies, represented in our membership and having Home Offices here: the Boston Mutual, the Columbian National, the John Hancock Mutual and the New England Mutual. All of us, deeply appreciative of the honor bestowed, have cheerfully co-operated in the effort to make this a pleasant, a memorable, a worth-while occasion.

I shall attempt no formal address. A few casual remarks, partly in reminiscence, partly in prophesy, and we shall pass on to the more important aspects of our program.

As I look upon this assembly and try to envision how it came into being and whither it is bound, I cannot but cast back for contrast to that first simple meeting of our Association, held on Memorial Day of 1890, fifty years ago, at the Buckingham Hotel in New York City. Thirty-four charter members, from twenty-seven companies, then comprised our Association. They were met ostensibly to consider,—with

what degree of gravity the record does not state,—a program of one paper: "The Influence on Longevity of the Use of Large Quantities of Beer." There were doubtless many aspects of that meeting which have never been recorded: the good fellowship; the plans, the dreams and hopes for the future of the Association; the discussion of ways and means to achieve its aims, to learn how to select life insurance risks, how to substitute for the insecurity of multiple personal opinions the certainty of life insurance medical knowledge.

It is beyond the powers of our imagination to assume that any member of that body could have had even an inkling of the tremendous role which the institution of life insurance was to play in the social and economic development of our nation over the next fifty years. No one of them could have envisioned the growth of the science of life insurance medicine which was to stem very largely from that simple beginning.

We are privileged to look back over the record, some of us even in memory, and note the steps in the growth of life insurance medicine made possible by friendly co-operation and joint action between companies, and particularly by that happy and momentous collaboration of the Actuarial Society of America and of our Association. We may enjoy and profit from the fruits of the labors of that splendid body of Medical Directors who have preceded us. Most of them, to our intense regret, have passed from our midst. We hold them in proud and grateful memory.

Their fidelity to the initial goal, their staunchness of purpose, their strenuous labor have borne this fruit: this present Association, of some 250 Medical Directors, representing some 100 life companies. Full well have they developed the science of life insurance medicine. Persistently have they encouraged those social and friendly relations between members which are the bed-rock of any persistent, successful organization. Outstanding has been their contribution to the advancement of the general interests of life insurance.

All of this has been achieved. It is accomplished fact. It

is our heritage, and as such endows us with definite responsibility. In our hands lie the future of our Association; the continued development of life insurance medicine; and,—a very fundamental factor,—the stabilization of the role of the Medical Department in the conduct of the institution of life insurance.

We may choose these alternatives. In the first place, we may lie back in the traces, content to utilize the knowledge already discovered for us, satisfied with a daily routine of executive Medical Department functions, with a medical selection which to a large extent has become automatic, carefully avoiding any approach to those realms in which inadequate knowledge makes selection uncertain. On the other hand we may venture forth to unknown fields, utilizing, to the full, current progress in medical knowledge, exercising a sensible judgment in adapting this for purposes of medical selection, and gradually extending the science of life insurance medicine to fields ever more remote.

As I see it, we cannot expect, to any considerable degree, a further development of the science of life insurance medicine by the methods which have prevailed in the past. Our knowledge as to the effect upon longevity of the common types of hazards is fairly complete. Repeated mortality investigations along these lines may in the future discover some changes, but relatively minor in degree and productive of no great alteration in our standards of selection, of no particular extension of the range of life insurance coverage.

In my opinion the life insurance Medical Department of the future,—if it is to justify its existence by exercising other than executive, clerical and supervisory functions,—must be alert to the constant progress in medical knowledge; quick to take advantage of its possible application to medical selection; cautiously willing to venture into obscure fields under the safeguard of cumulative mortality observations, thus by a restrained "trial and error" method, gradually extending the scope of life insurance medical science.

How shall Medical Departments keep abreast of current de-

velopments in medicine? The problems of medical selection cover every conceivable phase of human impairment. The competent Medical Director must of necessity have had a thorough general medical education and training at the outset of his career. His specialty, life insurance medicine, not only demands a particular knowledge of the problems of longevity, but also necessitates a general working knowledge of all the other numerous special fields of medicine. No Medical Director is able, by his own efforts, to maintain this diverse knowledge adequately attuned to medical progress. To be sure, in the staffs of the larger Medical Departments a numerous personnel facilitates the inclusion of physicians who have been trained in various specialties, who are able to keep abreast of current developments in their specialties. In the smaller Medical Departments this is not possible.

As a partial answer to this question, I believe that our annual meetings afford a means of refreshing the knowledge of the Medical Director. In fact it is now practically impossible to present here a program completely devoted to life insurance medical topics: a tacit admission of the practical stalemate which the development of life insurance medicine by means of the familiar methods of mortality investigation, has attained. During the last several years the trend toward the inclusion in our programs of discussions bearing upon the special fields of medicine has steadily increased. On this occasion, it is even more pronounced.

In formulating the program this year, the aim has been to present accurate information as to the present status of some phases of medicine in which the development of knowledge is so very rapid that it is impossible for most of us to keep pace; which confront us almost daily; with reference to which we have no statistical facts for guidance, being forced to rely entirely upon our own judgment and general medical knowledge.

The essayists who have so generously agreed to present these subjects can be relied upon as to the depth and accuracy

of their clinical knowledge and as to their willingness to attempt to answer the questions, which, from the point of view of life insurance medicine, their discussions may evoke. Full opportunity for questioning will be given you. I trust that you will not feel hesitant.

We, as Medical Directors, are indeed fortunate. We are an integral factor in the conduct of the institution of life insurance: an institution which stimulates and elicits the finest characteristics of mankind; which in these days of world-wide unrest, upheaval and injustice constitutes one of the most potent agencies of social and economic stabilization. It offers a field for the exercise of our peculiar training and talents, for their devotion to the benefit of our fellows, far more extensive than would be possible in any other range of activity. If, above the welter of our daily routine, we can occasionally catch the vision of the significance of our work, of our opportunities to be of real service to our fellow men, we shall become more useful, better Medical Directors, more content with life. After all, one of the keenest satisfactions of life is work, worth-while and well done.

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PRESIDENT FROST—We now proceed to the more important aspects of the program.

Donald Cragin needs no introduction to you men. He is, I think, one of the most alert Medical Directors whom it is my fortune to know.

He is here this morning to give you evidence of his alertness.

Without further introduction, I present Dr. Donald B. Cragin to you. He will discuss the subject of "Detection of Metallic Poisons and Some Types of Anemia: A Preliminary Report." Dr. Cragin!

DETECTION OF METALLIC POISONS AND SOME TYPES OF ANEMIA

A Preliminary Report

By Donald B. Cragin, M. D. and Sidney H. Roberts, M. A.

Aetna Life Insurance Company

In the last ten years enormous strides have been taken by industrial research workers, resulting in fabrication of many new materials and compounds. It behooves us as insurance carriers to inquire into the manufacture as well as the use of these new substances, particularly as to their chemical composition and the effect of constant exposure of workers to various elements which go into their making. The metallic poisons are with us always and exposure to metallic dusts and fumes has been difficult to underwrite quantitatively for life insurance because of the impracticability of laboratory tests. With the spread of popular insurance education and the subsequent increase in Group, Industrial, and Wholesale business, Life insurance has become available to the working man especially those of the lower salary groups who are exposed to the hazards of industry. Any attempt to underwrite by trade alone is unfair because modern industrial health programs and safety equipment make each exposure an individual study in itself; yet, with the possibility of present hazard and personal history of past disabilities, some method of rapid, accurate, quantitative determination is desirable to make a choice between the protected and unprotected worker in a hazardous occupation or in one suspected of being hazardous.

The principal troublesome metals are: lead, mercury, cadmium, arsenic, and selenium; with zinc, chromium, nickel, and copper in the offing. Some metals, such as mercury and cadmium, do not need to be quantitated since they do not appear in the urine of normal persons and hence their mere presence indicates exposure.

Formerly chemistry was practically our sole reliance in determining the metallic presence. However, any method that is concerned with amounts of the order of one part per million of a substance as soluble and ever present as, e. g., lead, has certain pitfalls in procedure which must be avoided, such as contamination and loss from repeated washing, impure "chemically pure" reagents, filter papers, soluble glassware, and small amounts of original sample to work with; vaporization from ashing in temperatures of over 300°C., colorimetric determination with back calculation (chromium to lead), etc.

About two years ago the spectrograph was brought to our attention. A brief description of its origin and construction follows:

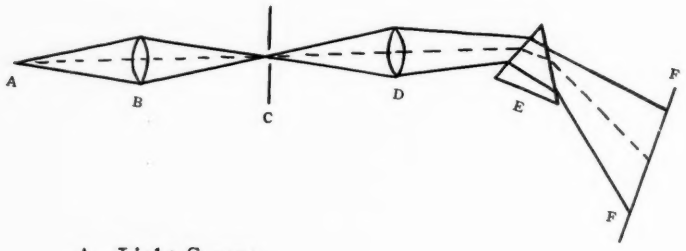
The essential characteristics of light may be explained by assuming that it is propagated as longitudinal waves. Measurements, therefore, are indicated by wave motion, that is, wave length, movement, and frequency. The speed of light has been shown to be constant in the range with which we are concerned.

In 1666, Sir Isaac Newton observed that if white light was passed through a prism it was dispersed in an orderly arrangement of colors, a rainbow or spectrum, proving that white light is composite. If the source is constant, these colors always occupy the same position, and in the case of pure white light, the same intensity.

In 1802 Wollaston, later followed by Frauenhofer (1814-7), employed a ray of sunlight passing through a narrow slit which made it function as a point of light in one dimension and observed that the spectrum was crossed by a number of black lines which were parallel to the slit. By placing a convex or condensing lens between the source of light and the slit, and a collimating lens between the slit and the prism, a better spectrum was obtained. Therefore, a spectrograph is an optical instrument consisting of (1) a lens system to concentrate the light from an incandescent sample to be analyzed, onto (2) a slit for making this light a narrow band, thence to (3) a collimator lens which makes the light from

the slit parallel before it strikes the dispersing prism or grating, and is carried thence to (4) a photographic plate. Prisms may be of glass or quartz, depending on the part of the spectrum which one wishes to investigate.

DIAGRAM OF SPECTROGRAPH



A – Light Source
B – Condensing Lens
C – Slit
D – Collimator Lens
E – Prism
F – Photographic Plate

Spectra may be classified in several different ways but first of all into: 1. Emission spectra and 2. Absorption spectra. The emission spectra are those resulting from the breaking up or dispersion of light admitted by the body under examination and are distinguished according to their general character as: (a) continuous spectra, (b) band spectra, and (c) line spectra. They may also be further classified as to the nature of excitation into (a) flame spectra, (b) arc spectra, (c) spark spectra.

Absorption spectra are those which result when a beam of light passes through a medium capable of absorbing, reducing in intensity, or extinguishing some of the rays before it enters the slit of the spectrograph.

The wave lengths may be: (a) the visible, which comprise only a small part of the spectrum, or (b) the invisible which include radio, Hertzian waves, infra red in the slow frequencies, and ultra violet, X-rays, Gamma, and cosmic rays in the high frequencies.

The unit of wave length measurement is called the Angstrom, after the Swedish physicist who devised it, and represents 1/6438.4696 of the wave length of the cadmium line. Each element has individual wave lengths or lines to which it adheres and which are never found elsewhere. Professor Harrison of Massachusetts Institute of Technology has recently compiled a most complete list of these wave lengths. When a sample is to be analyzed it is only necessary to decide which is the proper source of light and burn it (emission) or pass a beam of pure white light (absorption) through the compound, then read off the answer by the position and density of the photographic lines. The density is obtained by the use of a photoelectric cell and galvanometer comparing the original sample reading with a similar line from a sample of known concentration.

Increased accuracy of spectrographic methods has enabled physicists to measure every impurity in any metallic element. For example: under the ordinary flame spectrum one may recognize Barium to 1/2000 mgm., Potassium 1/3000 mgm., Calcium 1/50,000 mgm., and Sodium 1/14,000,000 mgm.

In the blood the method will trace out the amount of aluminum absorbed in a single meal containing only the usual amount of alum. It will also, when properly adjusted, reveal that the drug, separated from some toxicological material, for example, is not only morphine but that the quantity separated is even 1/100 part of a milligram or 1/6000 part of a grain.

It is used in detection of contamination in foods: by zinc, lead, copper, and tin.

The rare gases: Neon, Helium, Argon, were discovered by the spectrograph, as well as the elements: Thallium, Iridium, Germanium, Gallium, Rubidium, Caesium.

The scope of absorption spectroscopy includes: investigation of vitamins, alkaloids, and biological materials of toxicological concern such as: blood serum proteins, amino acids, bile pigments, etc. Particularly are we interested in the heme pigments and the compounds of haemoglobin; e. g. the haemochromogens, reduced haemoglobin, oxyhaemoglobin, carbohaemoglobin, methaemoglobin, sulph-haemoglobin, hematoporphyrin, corproporphyrin, cytochrome, urobilin, etc. These compounds all reveal definite absorption in certain positions of the spectrum and even with very small amounts of samples (less than 0.01 G. per liter) can be rapidly and accurately determined.

The changes in serum proteins in disease have been only meagerly investigated but a productive field awaits the worker.

Normal haemoglobin curves have been plotted. It now is necessary to obtain a series of blood samples in various hazardous exposures and learn the variations from the normal. We have started this plan of procedure.

Let us consider for a moment the subject of lead poisoning. This is of particular insurance interest because of the great number of industries all over the country where exposure to lead exists and because lead poisoning is not unknown in the home. Ingested lead is not as dangerous as lead absorbed through the skin or through the respiratory tract and so the dusty industries are the greatest offenders, with the wet processes a close second. Some of the typical hazardous industries are:

1. Ore handling and crushing
2. Lead casting
3. Alloy fabrication
4. The Pottery Trades
5. Glass making
6. The manufacture of pigments and glazes
7. Paint spraying
8. Printing
9. Gasoline mixing
10. Farm spraying

In the home lead exposure is usually suspected and discovered only when clinical symptoms develop. Then deleading treatment is started and continued until the patient is more or less symptom free. The life insurance underwriter wants to know how much lead the applicant is still carrying and how near the danger threshold of clinical poisoning he is. The most common means of acquiring lead poisoning in the home are:

1. Use of lead (pewter) dishes
2. Lead water pipes
3. Food contamination
4. Home medication (sugar of lead)
5. Home spray painting, etc.

As these exposures are all accidental and unlike the suspected exposures of industries using lead, any control is difficult. Very few people know the type of metal through which their water supply is piped!

The safe limit concentration of lead dust or fumes in air is "less than 1.5 milligrams per 10 cubic meters of air". The extreme dilution of this dust is best visualized by converting the lead to BB shot. One BB shot weighs about 30 mgm. and if in the form of dust would be dangerous in a room with a ten foot ceiling, 35 feet long, and 20 feet wide! The extreme danger to the workman occurs in the trades where lead oxide is shoveled in unventilated rooms; where dross is hand-skimmed from lead kettles; where lead oxide pigments are dry mixed; and where lead castings are handled in mass production.

As long as this lead is eliminated as rapidly as it is taken into the body, little or no harm is done. Lead elimination seems to cause little kidney damage in sub-dangerous amounts. As soon as the threshold limit is reached and lead is absorbed faster than it can be eliminated, it is deposited in the tissues, especially in the bones. This threshold limit is usually reached when the lead content of the urine is .1 mgm. per liter. A urine lead content of .2 mgm. per liter may or may not cause clinical symptoms but certainly shows a hazardous environ-

ment and potential plumbism. Clinical symptoms are usually present when the lead concentration reaches .3 mgm. per liter.

Insurance laboratory data is only of value when the information is furnished far enough in advance of clinical symptoms to enable measures to be taken to correct the exposure and hence lessen the severity of the crisis. When albumin, casts, blood, or hematoporphyrinuria occur, the poisoning is already well advanced. Basophilic stippling of red blood cells is not specifically found in cases of lead poison but is present in many other poisonings, such as benzene, arsenic, aniline, copper, or in secondary anemia, leukemia, malaria, etc. Some workers under known lead exposure do not show stippling at all; in others the stippling follows the clinical signs of lead absorption and toxemia rather than preceding them.

Chemical methods for detection and measurement of lead or mercury are tedious and require comparatively large initial samples for accuracy. Various chemical methods, although giving reproducible results, do not check each other because of the inherent losses due to the necessary manipulation of the different testing procedures. The use of a spectrographic method cuts manipulation to a bare minimum and, as the size of the sample does not vary the relative error. Thus it allows the use of very small samples if need be. Concentrations of lead in urine as low as one part in 100 million can easily be detected and one part in 10 million quantitated—and this in about one man-hour of work.

The advantages of the spectrograph are: small amounts of material, rapidity of analysis (one man-hour for a lead sample of urine), accuracy to a marked degree (third decimal point of a mgm.), and a graph as a record.

The disadvantages are the initial cost, though the machine is not expensive to run and is indestructible, and the absolute accuracy necessary in all manipulative processes. A technique as meticulous as that employed in aseptic surgery is essential to avoid contamination of samples. The method is so sensitive that if the operator's apparently clean and thoroughly rinsed fingers accidently touch the pure carbon electrodes, there is

sufficient minute contamination from the sodium in the soap previously used to result in the appearance of the sodium band on the spectroscopic plate.

So far we have done over 100 lead analyses and a considerable number of haemoglobins. We hope next year to present you with a detailed analysis of cases in which the spectrograph has been of value in underwriting and to further show its possibilities.

It seems to us that there are great possibilities in the instrument and after nearly two years our enthusiasm has not been dampened.

PRESIDENT FROST—Are there any questions you wish to ask Dr. Cragin? If not, we will proceed to the next paper on our program.

In the past, we have been privileged with communications from Dr. Rein. This year, we are again privileged. When Dr. Rein agreed to present his paper, he advised me that the title would be "The Significance of Positive Serological Examinations" but for some reason he has made up his mind that it is a pretty knotty problem, so he now changes his title to "Serology—A Problem." Dr. Rein!

SEROLOGY—A PROBLEM

By Charles R. Rein, M. D., New York City
Margaret G. Stephens and Marguerite LeMoine
Medical Division
Metropolitan Life Insurance Co.

Insurance underwriters have long had the problem of how to rate an applicant who voluntarily gives a history of syphilis. A more difficult problem is to detect applicants who conceal their history of syphilis or to discover those latent syphilitics who do not know they have the infection. Positive blood tests are sometimes the only evidence of syphilitic infection and are not infrequently the sole means of detecting syphilis in its latent and concealed stages. It is reasonable to expect a certain percentage of those seeking insurance to be syphilitic in the latent or asymptomatic stage, which in many instances can only be detected by positive blood tests. Since some companies impose strict physical examinations and require bloods tests for syphilis, while others are less strict and do not require blood tests, applicants who know they have syphilis may tend to apply to one of the latter companies.

For many years one of the criteria of a good risk is whether or not the treatment has been adequate. The difficulties in determining whether treatment is adequate are apparent. Even though the statements of the attending physician certifying to adequate treatment are true and accurate, there is always a reluctance to accept them unless they come from known specialists in this field or from hospitals or laboratories of high standing.

Insurance companies, therefore, should be especially interested in requiring a routine blood test for syphilis preceding the issuance of large policies in order to obtain a better selection of risks. If this were done thousands of people would discover that they have syphilis at a period when the late destructive stages of the disease may still be avoided. Such routine blood

testing would also serve as an important educational program and would give valuable statistical data on the prevalence of syphilis.

This paper will discuss some of the serologic tests and their application to life insurance and then discuss some of the problems of underwriters concerning the interpretations of the results of serologic testing.

In selecting a serologic test for routine insurance purposes, it is desirable to use one that not only possesses adequate specificity and sensitivity, but also that requires a small amount of blood, easily obtained, which may conveniently be sent to the Head Office or laboratory for examination.

The need for accurate information regarding the relative efficiency of the many serologic tests led to the formation of the Committee on Evaluation of Serodiagnostic Tests for Syphilis. This was accomplished by the U. S. Public Health Service in cooperation with the American Society of Clinical Pathologists. The initial study of this committee (1), (2) was to evaluate the several distinctive technical methods developed by American serologists. As a result of this study, five methods (Eagle, Hinton, Kahn, Kline, and Kolmer) were selected as the best tests, because of their practical adaptability, their wide-spread use, and their relatively high efficiency, i. e., specificity and sensitivity. The specificity of a test is determined by the percentage of false positive and doubtful reactions obtained in a large series of presumably *non-syphilitic* individuals, i. e., if one false positive reaction per hundred is obtained, the specificity of that particular test is 99 per cent. The sensitivity of a test is determined by the percentage of positive reactions obtained in a series of known syphilitic individuals. If 80 positive reactions per hundred are obtained, the sensitivity of that particular test is 80 per cent. Moore (3) points out that a serologic test should possess, as a minimum, 99 per cent specificity and 80 per cent sensitivity. These percentages should be considered the base line and tests conforming to the standards are satisfactory for diagnostic purposes.

Of the five methods selected by the Committee on Evaluation of Serodiagnostic Tests, the Metropolitan Life Insurance Company selected the Kline test in 1932, because in addition to its established efficiency, the test could be performed with a few drops of blood easily obtained from a simple finger puncture. The samples are collected in small, inexpensive capillary pipettes and are then mailed to the Home Office laboratory for testing. The detailed technique of collecting the samples and performing the test, as well as results obtained with the test at the Metropolitan Life Insurance Company, have been previously presented before this Association (4,5,6).

Descriptions of new or modified serologic tests for the detection of syphilis are constantly appearing in the literature. The Mazzini flocculation test (7) (8) has been evaluated in recent serologic studies and found to possess a high degree of sensitivity and specificity. It differs from the Kline tests in the type of antigen employed but it is very similar to the Kline tests in the technical details of performance. The Mazzini test can be done with a few drops of blood easily obtained from a finger puncture and is therefore especially applicable for insurance work. This test is now being investigated in the laboratories of the Metropolitan Life Insurance Company.

In a series of 2173 specimens there was an agreement of the Mazzini and Kline tests in 2079 sera (95.7 per cent). There was a disagreement in 94 sera (4.3 per cent). (See Table).

The definitions of this comparative study were: "Agreement", positive or negative by both tests; "Disagreement", positive by one test and negative by the other; and "Relative agreement", doubtful with the one and either positive or negative with the other test.

Commercially prepared Kline antigen and cholesterol solution for the Kline diagnostic test may be purchased already prepared and checked from the La Motte Chemical Products Company, McCormick Building, Baltimore, Maryland. The Mazzini cholesterinized antigen and buffered saline solution may be purchased already prepared and checked from the Eli Lilly and Company, Indianapolis, Indiana.

So much for the description of various tests. But even when the underwriter gets the serologic report, he is often handicapped by lack of other pertinent information. It might be helpful at this point to explain to some extent the difference

COMPARISON OF MAZZINI TEST
WITH KLINE DIAGNOSTIC TEST

	Sera	Percent
Total	2173	100.0
Agreement	2079	95.7
Absolute	1952	89.8
Relative	127	5.9
Disagreement	94	4.3
Positive Kline Negative Mazzini	70	3.2
Positive Mazzini Negative Kline	24	1.1

in the point of view of the clinician and the underwriter. In clinical practice, the physician will not make a diagnosis on a single positive blood test, but will re-examine the blood, carefully question the patient regarding a venereal history and previous treatment and then subject him to a thorough physical examination and special spinal fluid and cardiovascular examinations whenever indicated. As a result of all these data the physician is in a better position to establish a diagnosis of syphilis, and to determine the approximate duration and stage of the infection, which are important from a prognostic point of view.

The question of determining the adequacy of treatment in known treated syphilitics is very difficult even in private practice where the physician may expect full cooperation from the patient in regard to accurate history and thorough examinations. The Cooperative Clinics which were established by the U. S. Public Health Service, have set up certain data and criteria for determining adequate treatment in early, late, and latent syphilis.

To discuss in detail just what constitutes adequate treatment would take so long that only a brief definition will be given here. Adequacy of treatment will vary considerably with each individual patient and with the stage of the disease.

In early syphilis, adequate treatment should include a minimum of twenty injections of an accepted arsenical and twenty injections of a heavy metal (either bismuth or mercury) without rest periods. In latent asymptomatic syphilis, with negative cardiovascular and central nervous system findings, the treatment should be continued for a minimum of two years, depending on the age of the patient and his tolerance to the antisyphilitic medication. In either group, if the patient has received only a few injections of arsphenamine, regardless of the number of bismuth or mercury injections, the treatment must be considered inadequate.

The insurance underwriter seldom has opportunities and facilities to aid him in establishing a diagnosis of syphilis or deciding upon the efficacy of treatment but must underwrite the case on probabilities based on statistical frequency. Since most of our serologic work has been done with the Kline tests, only those results will be given. The Kline results will be referred to as negative, doubtful, weakly positive and strongly positive. A doubtful test refers to a one plus reaction; a weakly positive test is equivalent to a two plus or three plus reaction; and a strongly positive test is equivalent to a four plus reaction.

Last year, a chart was exhibited (see chart) to this Association showing the incidence of positive Kline reactions obtained in various groups of applicants for insurance, divided according to the reason for ordering a serologic test. In addition we now have the results of routine Kline tests on 13,000 Home Office employees at their annual physical examination.

The underwriter's problem is how to interpret a serologic report (1) when the applicant has a history of syphilis, (2) when the blood test is ordered because of signs or histories suspicious of syphilis; and (3) when a blood test is ordered routinely.

The first question is: *"What is the significance of a negative, doubtful, or positive serological report when the applicant has a history of syphilis?"*

The value of a negative report depends on the sensitivity and specificity of the test used. If the test is relatively insensitive, such as the Wassermann test with a plain alcoholic antigen, the reaction may be negative although the patient still has active syphilis. If, however, a more sensitive flocculation test is used, such as the Kline exclusion or Kahn presumptive test, a negative reaction is likely to indicate that the patient is "serologically cured." In clinical practice the patient would be considered "cured" only if he had received adequate antisyphilitic treatment, the very sensitive blood tests have been negative on at least three successive occasions, and thorough physical examinations including cardiovascular and spinal fluid examinations revealed no abnormalities. In insurance underwriting, a negative blood test and evidence of adequate treatment may be considered a probable "serologic cure" and the applicant rated accordingly. It must be emphasized, however, that it is possible for a patient or applicant with known syphilis to have positive spinal fluid or cardiovascular findings even though he may have a negative blood test.

A doubtful or positive reaction obtained in an applicant with a history of syphilis indicates that in all probability he still has syphilis. The clinician occasionally encounters syphilitics with persistent doubtful or positive blood tests, who have received adequate treatment, whose physical examinations reveal no indication of syphilis, and whose spinal fluid and cardiovascular examinations are negative. Such individuals may be considered Wassermann-fast or reagin-fast, and may live to an advanced age without further treatment.

Some insurance underwriters may have a tendency to rate syphilitic applicants according to the titre of their blood, on the assumption that the lower the titre, the better the prognosis and the more favorable the risk. Thus a syphilitic applicant with a doubtful blood test (plus one) is given a better rating than the syphilitic applicant with a weakly positive (two plus or three plus) or strongly positive (four plus) blood test. Such an interpretation is erroneous. Certain applicants with latent asymptomatic syphilis may have strongly positive

blood tests and still live to an advanced age, while other applicants with a syphilitic involvement of the heart, aorta or spinal cord, may have doubtful or weakly positive blood tests and yet be much graver risks. It is interesting to note our experiences among Home Office employees. In a series of seventy-seven (77) known syphilitics treated and untreated the following serologic results were obtained on their first routine serologic examination:

Strongly positive reaction	17	(22 per cent)
Weakly positive reaction	48	(62 per cent)
Doubtful reaction	12	(16 per cent)

It is known that among those with doubtful reactions there are almost as many individuals with syphilis of the cardiovascular or central nervous system as in the strongly positive group. Therefore I stress again that the reagin titre of individuals with a history of syphilis is not significant, and all such applicants with doubtful, weakly or strongly positive serology, should be similarly rated.

The second question is: *"What is the significance of the serologic reports when blood tests are ordered because of signs of histories suspicious of syphilis?"* Doubtful or weakly positive reactions obtained in applicants with irregular pupils, abnormal reflexes or other signs of syphilis are presumptive evidence that these abnormalities are caused by syphilis. A positive or doubtful serologic reaction in such cases would, of course, be considered syphilis for insurance underwriting. In this regard it is interesting to note the serologic results obtained in special groups of applicants examined at the Home Office. In a series of 7788 applicants whose blood was tested routinely only 2.6 per cent had doubtful or positive reactions. When blood tests were requested in a group of 181 applicants because the pupillary reaction to light or accommodation was absent or sluggish, 31.5 per cent had doubtful or positive reactions. In a group of 76 applicants with uneven or irregular pupils, 17.1 per cent had doubtful or positive reactions; and in a group of 84 applicants with absent or diminished patellar reflexes, 8.3

per cent had doubtful or positive reactions. In clinical practice such individuals would be subject to a thorough examination before a definite diagnosis of syphilis would be established. In insurance work, however, where such thorough examinations are not always possible or feasible, all applicants with abnormal reflexes associated with doubtful or positive blood tests may be considered syphilitic and rated accordingly.

The third question is: *"What is the significance of serologic reports from applicants who have routine blood tests, or where such tests are requested because of the amount of insurance applied for?"*

The results of the routine serologic examinations of the employees of the Metropolitan Life Insurance may be of guidance to the insurance underwriter. These employees were first examined at the Home Office by the finger blood method. If the test was doubtful or positive, the individual was re-tested within a few days or weeks. If the doubtful or positive reactions persisted, the individual was referred to a syphilologist for further examination. This consisted of taking venous blood from the arm for rechecking the Kline tests and doing other serologic tests for syphilis. In addition, the employee was carefully questioned regarding a venereal history. It was not always possible to do a complete physical examination and seldom feasible to do a spinal fluid examination. Yet, in spite of these inadequate examinations, some interesting and pertinent information was obtained.

The number of employees who had doubtful or positive reactions in one or both years amounted to 304, an average of 1.42 per cent per year. About one-quarter of them were diagnosed as syphilitic, an incidence of less than one-half of one per cent each year. The others were considered to have non-specific serology, because they presented no clinical evidence of syphilis and they had no knowledge of a specific infection.

Among those who were diagnosed as syphilitic, a few were known to have had treatment for syphilis before the routine Kline tests were instituted; about one-half of them had been

detected by routine tests previously done on certain small groups. Some of those who showed a positive reaction on routine testing but had no history of syphilis on their medical record with the Metropolitan, admitted to the syphilologist that they were aware of the infection. Some had no knowledge of infection. Thus, the syphilitic group, as a whole is comparable to applicants for insurance in that their syphilis was either unrecorded on their medical record or unknown to themselves.

The experience in this group may be taken as a guide for underwriting individuals who do not admit a history of syphilis. The findings for the males were so different from those for the females that the two sexes should be considered differently for underwriting. Among the men, when the Kline test was three plus or four plus, a diagnosis of syphilis was established in nine out of ten cases; when the Kline test was two plus, a syphilitic infection was proved in six out of ten cases; and when the Kline test was one plus, syphilis was proved in three out of ten cases.

Among the women the results were quite different. Syphilis was diagnosed in about four out of ten having three plus or four plus reactions; in about 1.5 out of ten having two plus reaction, and less than one out of ten having one plus reaction.

A doubtful or positive reaction in a person who denies a syphilitic infection and who shows no clinical evidence of the disease on examination, presents a very delicate problem for the physician and for insurance companies. This raises the question of what to tell the applicant's physician if he inquires about the cause of rating or declining the application because of a serologic finding. The Metropolitan Life is now sending the following statement to the inquiring physician:

> "The serologic report indicates an unusual condition, though not necessarily pathological. If after further clinical study you feel that no significant abnormality is present, will you please let us know."

If the physician, on further examination believes that the applicant does not have syphilis, and that the doubtful or positive blood tests obtained by the Metropolitan Life Insurance Company were non-specific, the application may be reconsidered.

In addition to the data on proven specific reactions, the experience with presumed non-specific reactions may be of interest. Among the males the incidence of non-specific reactions was about the same at each five year age period. Among the females, the highest incidence of non-specific reactions occurred in the youngest group under age 20 and decreased rapidly with advancing age.

It was thought that the increased number of non-specific reactions occurring in the younger females might be due to menstrual or endocrine disturbances. A series of females with presumably non-specific reactions had blood tests on the first or second day of their menstrual period and again exactly two weeks later. It was found that there was no appreciable difference in the number of non-specific reactions or in the reagin titre in the blood specimens collected during and between the menstrual periods.

CONCLUSION

1. Insurance companies should be encouraged to do routine serologic examinations to detect syphilis in applicants.

2. The type of serologic procedure selected should be one that has been proven to possess adequate sensitivity and specificity, and one that can be done with a few drops of blood easily obtained from a finger puncture, and which can be conveniently mailed to a central laboratory for examination. The Kline and Mazzini flocculation tests have proven to be especially applicable for insurance work in our laboratory.

3. Doubtful and positive reactions obtained in applicants who admit a history of syphilis, should be accepted as evidence that the individual still has syphilis and should be rated accordingly.

4. The titre of blood of persons with a history of syphilis is not significant. Underwriters, therefore, should not rate such syphilitic applicants according to the degree of positivity of the serologic reaction.

5. A doubtful or positive reaction in an applicant who has signs or a history suspicious of syphilis is evidence that the abnormalities may be caused by syphilis.

6. A doubtful or positive blood test obtained in an applicant who denies a syphilitic infection and shows no clinical evidence of the disease presents a delicate problem. The experience obtained from the Metropolitan Life Insurance Company employees is quoted as guide to the insurance underwriter.

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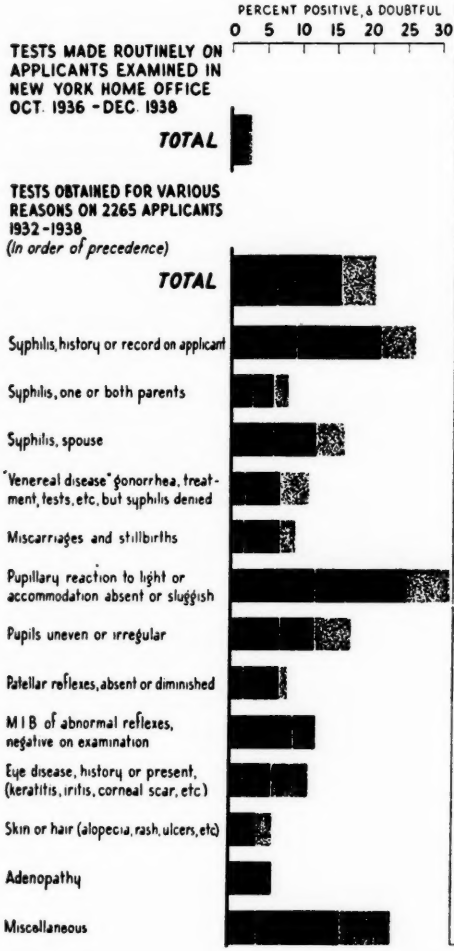
RESULTS OF KLINE TESTS
ON APPLICANTS FOR LIFE INSURANCE
METROPOLITAN LIFE INSURANCE COMPANY



	PERSONS TESTED	PERCENT		
		STRONGLY POSITIVE ++++	WEAKLY POSITIVE +++, ++	DOUBTFUL +
TESTS MADE ROUTINELY ON APPLICANTS EXAMINED IN NEW YORK HOME OFFICE OCT. 1936 - DEC. 1938				
TOTAL	7788	2.2		0.4
TESTS OBTAINED FOR VARIOUS REASONS ON 2265 APPLICANTS 1932-1938 *(In order of precedence)*				
TOTAL	2265	6.4	9.1	4.6
Syphilis, history or record on applicant	1021	8.9	12.2	5.2
Syphilis, one or both parents	113	2.6	2.7	1.8
Syphilis, spouse	49	6.1	6.1	4.1
"Venereal disease" gonorrhea, treatment, tests, etc, but syphilis denied	98	2.0	5.1	4.1
Miscarriages and stillbirths	238	1.7	5.4	2.0
Pupillary reaction to light or accommodation absent or sluggish	181	12.2	13.3	6.1
Pupils uneven or irregular	76	6.6	5.3	5.3
Patellar reflexes, absent or diminished	84	2.4	4.8	1.2
M I B of abnormal reflexes, negative on examination	33	9.1	3.0	–
Eye disease, history or present, (keratitis, iritis, corneal scar, etc)	129	2.3	3.9	5.4
Skin or hair (alopecia, rash, ulcers, etc)	58	3.5	–	1.7
Adenopathy	32	–	6.2	–
Miscellaneous	153	3.9	12.4	7.2

PRESIDENT FROST—In arranging for the discussion of Dr. Rein's most interesting and provocative paper, it occurred apparently to Dr. Rein and myself that a discussion from the point of view of a clinical expert in these matters, and I will say another clinical expert in these matters, would be of benefit to us. Dr. Rein himself suggested an expert here in Boston, whom I have known for many years, and for whom I have the highest regard. I know that he is one of the outstanding men in Boston as to his depth of knowledge of the subject which we have under discussion.

This man, Gentlemen, is Dr. John Godwin Downing, and it gives me pleasure to present him to you to discuss Dr. Rein's paper. Dr. Downing!

DR. DOWNING—Mr. Chairman and Members, I appreciate your invitation to come here for discussion of Dr. Rein's excellent paper. He is to be congratulated on his thorough exposition of the problem of serology. Routine blood tests, detailed physical examinations, and careful histories will detect most cases of syphilis. However in a life insurance examination, history is so inaccurate, so unreliable that I am convinced that the elimination of the question concerning it would entail no financial loss and cause less injustice. The only credible affirmation is made by a person adequately treated, with resulting negative serology, spinal fluid, and physical examination. He, confident of his apparent cure, looks forward to a normal span of life, only to be confounded when underwriters disagree with this view-point and offer him a policy with a greatly increased premium. I believe that this applicant, conscientious enough to undergo prolonged treatment, has learned the value of maintaining a normal, healthy routine, and will enjoy better health than many of his contemporaries.

Doctor Rein stresses what constitutes a desirable serologic test and cites the Kline test which has proven its efficiency. In Massachusetts we are justly proud of the Hinton test with its evaluated sensitivity of 88.8% and specificity of 100%.

With the Davies modification it is now adaptable to small quantities of blood and results show a sensitivity of 91.9% and specificity of 100%.(1)

Persistent doubtful or positive reactions bother clinicians as well as underwriters and it is impossible for either to evaluate properly these cases. Although I agree with Doctor Rein as to their probable longevity and assure these patients that excessive treatment is unnecessary, still I do keep them under yearly observation. The value of routine serologic tests is exemplified by his examinations of the employees of the Metropolitan Life Insurance Company. His figures closely agree with my one and one-half per cent positive reactions found among industrial workers referred to my office, for consultations regarding their cutaneous eruptions, by insurers.

In industrial work the finding of a positive reaction is always embarrassing. Reporting it to the insurer convinces him that the eruption, no matter what it may be, must be syphilis. It may start a rumor which will reach the employer with subsequent discharge despite the dermatologist's protestations of non-infectiousness. Fellow workers may learn of the patient's condition and this knowledge will precede him in his search for employment. If the case doesn't warrant need of notifying the insurer, it is better to inform the worker of the result of the test and make sure he reports to his family physician for further examination, and treatment if necessary. Recent education has been valuable to both employer and employee. Industry now realizes the cost of syphilis, the importance of knowing who among the employees are syphilitic, and what should be done for them. One traumatic injury to a worker with a non-treated syphilis may cost more than a year's routine serologic tests on all employees.

I do not believe that a positive blood test should keep any worker with a negative physical examination from any type

(1) 1. Venereal Disease Information, U. S. Public Health Service, Jan. 1937: Vol. 18: No. 1, p. 4. Efficiency of State and Local Laboratories, Serodiagnosis of Syphilis.

of work. Each case should be studied and treated accordingly. If he has open lesions he should not be allowed to work until these are healed; if he has cardiovascular or neurological damage he is not able to work except in selected cases.

An important factor in our national defence will be the maintaining of the efficiency of our industrial workers. Syphilis, one of the greatest menaces in industry, can be reduced by periodic serologic examinations and early treatment.

PRESIDENT FROST—It seemed desirable to have an additional discussant of this paper from the point of view of the life insurance Medical Department. For this purpose, Dr. Charles K. Wallace, Assistant in Hygiene of the Travelers' Insurance Company, has agreed to contribute. Dr. Wallace!

DR. WALLACE—Mr. President and Gentlemen, it is evident to one who hears this paper that Dr. Rein and his associates have taken great pains to obtain their data and that they have made a thorough study of the problem.

It is not always easy to make a diagnosis of syphilis or to exclude it. For Life Insurance Underwriting purposes it is a particular advantage to have at least two different kinds of tests done on each specimen.

It is expected that a similar study to that made by the United States Public Health Service and American Society of Clinical Pathologists will soon be started in the Connecticut State Approved Laboratories. We will know more about the specificity and sensitivity of the tests as we do them in each Laboratory. We will welcome this study.

The great number of tests being done by Insurance Laboratories on applicants for insurance, on applicants for employment and on employees will have a tendency to decrease the incidence of syphilis directly and have a good influence on public opinion in the work against syphilis.

Three thousand five hundred and six cases had both the

Kline Test and the Hinton Test (capillary method modified by Davies) done in our Laboratory. There was agreement in thirty-eight cases where there was some reaction other than negative in both Kline and Hinton Tests. There were Kline reactions other than definitely negative in eighty-five cases who had definitely negative Hinton Tests, where the Kline was definitely negative. There were nineteen people whose blood gave strongly positive Kline and Hinton Tests.

It is difficult to compare one's results with that of another Laboratory even though the same test is used. One worker may call it one plus, another might call it doubtful, so I think it is not important in this discussion to compare results. The figures do show how one test seems to give a positive reaction more frequently than the other test.

It seems to me that it is important to study when possible a case showing a positive reaction. However, unless there is clinical evidence or history, I think one should not be too apt to refuse an applicant unless at least two different kinds of tests are positive.

Sometimes underwriting these cases may seem difficult but it is less difficult than formerly. We know more about the sensitivity and the specificity of the tests than before the country-wide studies were made. We shall know more when more and more studies shall have been made.

With Dr. Rein's conclusions I am very much in agreement and I am pleased to have had the opportunity to discuss the paper.

PRESIDENT FROST—We have a very few moments for discussion. Would any one care to discuss Dr. Rein's paper?

DR. EXTON—I should like to say that Dr. Rein would have done better if he had stuck to his original title, with the headache, because from the nature of the test, I think you will always have a headache with it. The test is based on discovering a small quantity of substance evolved between the tissues of the spirochete by non-specific antigen and the tests that have been

produced by skilful and enthusiastic serologists have been, as it were, balanced between specificity on the one hand and sensitivity on the other hand.

Any one of them would be able to make his tests more specific or less sensitive, according as he does his manipulations, and the result is that if you use two tests, you always have more headaches than if you use one test.

I should like to make a further point and agree and reemphasize what Dr. Rein said about the foolishness of using the so-called quantitative, doubtful one, two, three, four, plus, or whatever you call them; if there is any reaction at all and the reaction is specific, it indicates the presence of syphilis, and when it comes to rating or taking these cases, whether it is a large or small amount, it really doesn't make much difference if they have syphilis.

Now, there is a question I would like to ask Dr. Rein, about these slight and doubtful reactions. I wonder if he has any data on the persistence of these reactions in more than one examination. For instance, I have been under the impression that if you got a doubtful reaction and it persisted, that indicated syphilis. I should like to know if he has had any experience along that line.

PRESIDENT FROST—Is there any further discussion of Dr. Rein's paper? If not, I am sure Dr. Rein has a few more words to say to us now.

DR. REIN—I want to thank the discussants for the excellent and flattering discussions.

I agree with Dr. Downing about patients with the four plus blood tests in the latent stage, who may go along for a long time and not need any more treatment. In clinical practice, we have the opportunity to watch these patients, and we can do something about it, if anything goes wrong. But, in insurance work, you don't have the opportunity; once you insure him, you're finished. If something goes wrong, you are out of luck.

I am also happy that he cautioned you about the five-day treatment. It is very new, and, of course, we who are from

New York are very much prejudiced in its favor, maybe because of home and environmental honors bestowed upon us. But, it is new and should not be accepted as yet. New studies are being started next week in New York on the problem. We hope it may answer a problem. We know, definitely, that it answers problems in the prisons, where ninety out of every one hundred women arrested are syphilitic. We know that by the five-day treatment, even if we don't cure them, if they go out and apply their trade of prostitution, they may affect less people. We hope that some day we will have the answer to the five-day treatment.

Dr. Wallace stressed a point about doing two tests for syphilis. I think we have gotten past that stage today. I think the government has established once and for all that any one of these tests is adequate.

I agree with Dr. Exton about the headache; if I keep on I know I will end up by having a stroke!

As far as persistent doubtfuls in our non-specific and specific cases are concerned, it is my impression that syphilitic patients with a specific doubtful may persist a long time. Dr. Fellows has given me the opportunity of watching patients at the Metropolitan Life Company who are doubtful; even non-specific doubtfuls may persist. If it is due to some metabolic disorder, it may persist for a long time, and such patients may continue to produce the doubtful or positive reactions as long as this disorder exists.

So that we can't say for sure how long a non-specific doubtful will persist.

The Metropolitan Life has a little kit which they send out to the examining physician. In that kit, there is a self-addressed blank, to mail back to the company, also a little equipment, including a capillary pipette. All he has to do is collect two or three of these to the full point and put them in the container and send them back. If these are filled adequately, we have enough to do four or five tests and repeat the tests, if necessary. I heard one physician say, one day,

that he seemed to get the blood over the box and the table, but I think those cases are extremely rare. Any doctor who has once taken a blood count should be capable of taking the blood from the finger for one of these tubes. He may have some difficulty in getting into the arm, but he certainly should have no difficulty in collecting enough blood in the capillary pipettes.

I am sure that Dr. Fellows and the members from the Metropolitan Life would be glad to send you the data.

I want to take this opportunity to thank you for the opportunity of presenting this paper.

PRESIDENT FROST—Dr. Rein, we all extend to you our thanks, not only for your excellent paper, but for its admirable presentation. We trust you will extend our thanks to your associates, also.

PRESIDENT FROST—With respect to the next paper, I don't suppose that I am particularly different from the majority of you men here. When the conversation drifts to vitamins, and when your friends ask you what this and that vitamin does and what it does not, when you are faced with the multiplicity of advertisements of this, that and the other publication of various products with vitamins and the glowing claims as to what they will do, I find myself in somewhat of a morass of uncertainty. Every once in a while, I go over these vitamins, and I think I have them memorized, but then in three or four days, I have forgotten all about them. The various deficiency diseases come up, occasionally, in medical selection work, and sometimes I wonder what I shall do with this, that and the other type of deficiency disease. I appreciate my lack of knowledge in that particular phase of medicine. That is why we have our next paper on the program this morning.

In casting about for an individual to present a discussion on this subject, I, naturally, because I am a confirmed Bostonian, resorted to our local talent, and I think we have as good as there is anywhere. Therefore, I have asked Dr. Maurice B. Strauss to present this subject to you.

Dr. Strauss is a graduate of Johns Hopkins, 1928, and is now Associate in Medicine at the Harvard Medical School; he is on the staff of the Boston City Hospital, the Thorndike Memorial Laboratory, and he has numerous appointments in the City here, but the essential fact is that he is thoroughly versed in these abstruse matters—at least they are abstruse to me—with reference to disorders of the nutrition deficiency diseases, lack of vitamins, etc. Dr. Strauss has generously agreed to come and enlighten us on this subject of "Disorders Due to Nutritional Deficiency." Dr. Strauss!

DISORDERS DUE TO NUTRITIONAL DEFICIENCY

By Dr. Maurice B. Strauss

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(From the Thorndike Memorial Laboratory, Second and Fourth Medical Services (Harvard), Boston City Hospital, and the Department of Medicine, Harvard Medical School, Boston, Massachusetts.)

INTRODUCTION

The reaction of the medical profession to anything new has been graphically illustrated by Davis [1] (Chart 1). A period of study and investigation generally accompanied by scepticism is followed by over-enthusiasm and credulity, ending in gross abuse. Disillusionment results in over-correction. Only at long last does sane appraisal result in stability.

In the case of a therapeutic agent such as sulfanilamide the major portion of the curve has been run in a few years. We are already on the descending limb of caution and decreased use. With nutritional deficiency disorders the curve has been prolonged. Beginning with Eijkmann in 1897, or even earlier, the period of investigation unaccompanied by over-enthusiastic acceptance extended over at least three decades. Ten years ago, although scurvy and rickets were recognized as nutritional deficiency disorders, the controversy still raged as to whether Oriental beriberi was an infectious disease, and its existence in this part of the world was unheard of. In 1934 McLester [2] indicated his belief that contagion played a role in the etiology of pellagra, and this in spite of Goldberger's conclusive demonstration that the syndrome was one due to nutritional deficiency. Four years later, in 1938, it was estimated that 100 millions of dollars were being spent annually in the

United States for vitamin preparations! There are few diseases known to man today for which vitamin deficiency has not been offered as an important etiologic factor. These extend from poliomyelitis and the neuritis of leprosy on the one hand to rheumatic fever and spontaneous abortion on the other.

Certain types of fallacious reasoning appear so very commonly in the literature dealing with human nutrition that it is worth while to comment upon them. Now, it is apparent that a lack of quinine is not the cause of malaria, although this chemical certainly cures the majority of cases of the disease, nor is it thought that bronchial asthma, relieved by adrenalin, results from a lack or deficiency of this hormone. On the other hand, should any disorder be relieved following the administration of a vitamin or vitamin product, it is immediately concluded that the disorder is due to a lack of that substance.

There is a second type of fallacious reasoning that has recently been commented on in connection with the use of the biophotometer. This type of fallacy may be illustrated in connection with a consideration of red blood cell counts:

If a single specimen from an individual whose blood actually contains 5,000,000 cells per cu. mm. is divided into a thousand samples and an independent erythrocyte count is made in the usual way on each sample, practically all the counts will be found to fall between 3,800,000 and 6,200,000 cells per cu. mm. with a mean at 5,000,000. The count will be distributed with close approximation according to the gaussian curve ("normal" curve, Chart II) with a standard deviation of 390,000 cells per cu. mm. One hundred and sixty-six of the counts will be below 4,610,000; twenty-five of the counts will be below 4,220,000. If now we make new independent counts from the 166 samples which showed these lower counts, they will again fall according to a gaussian distribution with a mean of 5,000,000 and a standard deviation of 390,000. Only 27 will be below 4,610,000. The remaining 139 will have "improved". Should we have recounted only the 25 samples on which the first count was below 4,220,000 we would find that 24 of the

25 will now be higher. All the counts of course are supposed to have been made from samples from a single homogeneous specimen of blood in which the actual count was 5,000,000 per cu. mm.

This centripetal tendency of the extremes of any such series to approach the mean on re-examination has led to many alleged beneficial results of specific therapy.

A third type of error is the assumption that correlation is synonymous with casualty. Although it can be shown that the decline in typhoid mortality in Massachusetts closely parallels the rise in the number of telephones in use (Chart III), it is hardly believed that the possession of a telephone protects against typhoid.

Lastly, the natural tendency of many disorders to undergo spontaneous improvement regardless of treatment is commonly overlooked.

I shall now endeavor to summarize for you the *relatively well established nutritional deficiency disorders in man.*

Iron deficiency is probably the most prevalent of all nutritional deficiency disorders. It manifests itself in the form of hypochromic anemia—a condition easily detectable with simple apparatus—and is to be found in at least 10 per cent of the population. In the underprivileged, during infancy, at puberty and in pregnancy its incidence rises greatly. Iron deficiency rarely if ever arises solely from an inadequate intake of this substance. More often a dietary lack is associated with an increased requirement, as in periods of rapid growth (infancy, puberty), during pregnancy (blood building material for the foetus), in the presence of blood loss (normal menstruation, menorrhagia, bleeding hemorrhoids, epistaxis, gastro-intestinal tract disease, hookworm infestation) and not infrequently with impaired iron absorption from the alimentary tract (gastric anacidity, colitis, sprue).

The symptoms of iron deficiency arise primarily from the lack of blood. Their severity varies with the degree of anemia. Koilonychia and atrophy of the tongue occasionally

seen in iron deficiency anemias have not been proved to be the result of lack of iron, although they generally respond to iron therapy. Iron should practically always be administered by mouth in inorganic form. The extremely inexpensive ferrous sulphate is the form of choice in most instances.

Protein of good biologic value is of great importance in human nutrition. Man is not fundamentally an herbivorous animal. Although he may secure a varied enough and adequate protein intake from plant products, extreme care is necessary in selection. Animal products on the other hand insure a varied intake if the quantities are sufficient. A few decades ago the minimum protein requirement was considered to be 1.0 gm. per kilo of body weight or roughly 60 gm. for an average size adult. Today that figure has been raised to 100 or more gm., with even higher figures for periods of growth, pregnancy and great physical activity. A lack of protein may exist for a considerable period, during which time reserve stores are being depleted, before a lowering of the plasma protein values leads to the development of edema. Minor degrees of deficiency, insufficient to produce edema in the otherwise normal subject, may be a contributing factor in patients with heart disease, anemia and other disorders.

In earlier days the liberal use of protein was considered harmful in hypertension, kidney disease, liver disease and pregnancy. Today we realize that it is in just those conditions that an increased protein intake may be particularly desirable.

Pernicious anemia is a nutritional deficiency disorder of a type but recently recognized. Although it may occasionally arise as a result solely of an improper food intake, it most generally occurs because a gastric secretory defect prevents the elaboration from a food factor of the material necessary for the maturation of red blood cells, and may thus be considered as a "conditioned" deficiency disorder. Formerly a fatal disease, it now does not in any way decrease life expectancy as long as adequate treatment is carried out. Not a single

patient out of over 500 treated by my colleagues and myself has died of pernicious anemia while under treatment, once remission has been secured. Lacking police powers, however, we cannot compel certain patients who have remained in perfect health for years, to continue what may at times seem like unnecessary therapy.

Vitamin A. A deficiency of this material leads to keratinizing metaplasia of epithelium and to abnormally slow resynthesis of visual purple (rhodopsin) in the red cells of the retina. The former is responsible for xerophthalmia and keratomalacia. Conjunctival dryness and photophobia may be early manifestations. Follicular hyperkeratosis of the skin is frequently seen in states of impaired nutrition and has been considered due to a lack of vitamin A, although more recent evidence suggest that other food factors are involved. Nutritional night blindness results from the faulty resynthesis of rhodopsin. Other causes of impaired dark adaption are not uncommon; significant day to day variation occurs in biophotometer readings.

There is no evidence that vitamin A deficiency plays a significant role in the pathogenesis of the common cold, sinusitis, otitis, bronchitis or urinary lithiasis.

Vitamin B_1 deficiency in man involves predominantly the nervous and circulatory systems. As yet there is no evidence that thiamine hydrochloride is the material involved in the neural changes. In the northern United States, evidences of vitamin B_1 deficiency occur chiefly in chronic alcohol addicts, pregnant women who have had extensive vomiting, patients with diarrhea, ulcer, surgical resection of the intestines, thyrotoxicosis, prolonged febrile sepsis and diabetes as well as in food cranks. Anorexia, nausea and impaired growth are early manifestations of vitamin B_1 deficiency. These symptoms are however common in so many disorders that their presence has no diagnostic value. The chief neural lesion of vitamin B_1 deficiency is a characteristic polyneuritis clinically differentiable from other polyneuritides. There is suggestive evidence that both the mental changes of Korsa-

kow's syndrome and encephalopathy of Wernicke's syndrome are the result of nutritional deficiency, probably involving vitamin B_1 or some portion of the vitamin B complex. It is to be emphasized that as used here the term vitamin B_1 refers to the antineuritic dietary factor and necessarily to thiamine hydrochloride. The cardiovascular manifestations of vitamin B_1 deficiency do not comprise as clear-cut a clinical syndrome as the neural lesions. In the presence of the latter however, cardiovascular signs may be suspected to be of nutritional origin. Signs of congestive failure, in the absence of organic heart disease, particularly when associated with a rapid circulation time, should suggest the possibility of nutritional deficiency.

There is as yet no evidence that vitamin B_1 deficiency is involved in the neuritides of leprosy, diphtheria, lead and other toxic poisonings, subacute combined degeneration of the spinal cord, multiple sclerosis, Bell's palsy, trigeminal neuralgia, poliomyelitis, chorea, tabes dorsalis, or herpes zoster in spite of claims to such effect.

Pellagra is a syndrome of frequent occurrence in the South and of less common occurrence in the North and then only in the same type of individual noted above who is prone to develop vitamin B_1 deficiency. There appears to be no doubt that the syndrome of pellagra results primarily from nutritional deficiency. The components of this syndrome, not all of which are generally present in any one case, include dermatitis, cheilitis, keratitis, glossitis, stomatitis, diarrhoea, spinal cord degeneration, polyneuritis, dementia, cardiovascular disorders and anemia. The dermatitis appears to be due to a lack of nicotinic acid plus irritation from actinic rays, friction or bodily secretions. The oral lesions, diarrhea and dementia respond to nicotinic acid therapy. The keratitis and cheilitis are probably due to riboflavin deficiency. The polyneuritis and cardiovascular manifestations are presumably due to a lack of vitamin B_1. The spinal cord degeneration and anemia are no doubt nutritional in origin, but what factors are involved remains in doubt.

Nicotinic acid is a pharmacologically active substance and in certain dosages produces marked vasodilatation. Its amide, known as coramine, has been marketed for years as a cardiovascular stimulant. Such actions occur in normal man not suffering from nicotinic acid deficiency and must be sharply differentiated from effects produced when the substances act to supply a lack. The action of nicotinic acid in delirium tremens and certain cases of coma probably falls in the first classification. *Pantothenic acid* and *pyridoxin* are recently isolated fractions of the vitamin B complex, the latter being synonymous with vitamin B_6. Their importance in human nutrition has not been ascertained. No syndromes due to their lack have been established. No therapeutic use of either rests on sufficient evidence.

Riboflavin (vitamin B_2) deficiency has been shown to result in changes mentioned above. Redness and desquamation at the mucocutaneous junction of the lips, followed by ulceration, occurs. Macerated fissures are found at the corners of the mouth. The tongue is said to turn a magenta hue different from the scarlet stomatitis of nicotinic acid deficiency. Whether roughening of the skin about the nose with plugging of the pores is due to riboflavin deficiency is uncertain. The ocular manifestations of riboflavin deficiency commence with proliferation and engorgement of the limbic plexus, followed by superficial vascularization of the cornea and interstitial keratitis. Photophobia, dimness of vision and actual impairment of vision result.

Vitamin C deficiency leads to scurvy, a disorder characterized by a lack of intercellular cement substance. It is common in infants subsisting wholely on boiled milk formulas, in adults on diets restricted because of ulcer, colitis, etc., and in individuals who because of economic circumstances or food fads fail to eat fruit or vegetables. Petechiae and larger extravasations of blood occur spontaneously or after minor trauma in the skin, under mucous membranes, subperiosteally, under serous surfaces and from the gastro-intestinal tract. The

gums, when teeth are present, and particularly when they are in poor condition, become swollen and boggy and bleed easily. Teeth may loosen. Wounds fail to heal properly.

There is no evidence that a lack of ascorbic acid results in anemia. Its use in hemorrhagic disorders other than scurvy is without effect, as is its use in other dental disorders. There is no evidence that a lack of ascorbic acid leads to cataract, leukemia, hyperthyroidism, peptic ulcer, rheumatic fever or rheumatoid arthritis although such claims have been made.

Rickets and osteomalacia result from an imbalance of calcium and phosphorus metabolism which is largely due *in man* to a lack of vitamin D. These disorders are so well understood that they will not be discussed. Certain forms of vitamin D appear valuable in the treatment of parathyreoprival states with tetany and low blood calcium. The value of vitamin D in rheumatoid and hypertrophic arthritis has not been established.

Vitamin E (alpha-tocopherol) is essential for normal fertility in rodents. Its lack is also associated with lesions of a degenerative type in the musculature of these animals. There is however no satisfactory evidence that vitamin E deficiency plays a role in human infertility or spontaneous abortion or that its therapeutic use is beneficial in these conditions. The published reports of its use in muscular dystrophies and amyotrophic lateral sclerosis are very unconvincing.

Vitamin K is outstanding among the recently isolated nutritional substances by virtue of the fact that few unproved claims have been made for it. In its absence the prothrombin content of the blood diminishes. When the prothrombin is reduced to 20 to 30 per cent of normal, hemorrhagic manifestations appear. Prothrombin deficiency seems to be the largest single etiologic factor in hemorrhagic disease of the newborn and in the bleeding tendency present in obstructive jaundice, biliary fistulae, sprue, ulcerative colitis, coeliac disease and other disorders of intestinal absorption. Vitamin K is apparently not absorbed from the intestinal tract in the

absence of bile salts. A synthetic substance, 2 methyl-1, 4-naphthoquinone, having marked vitamin K activity is now available and has given dramatic results in prothrombin defi-

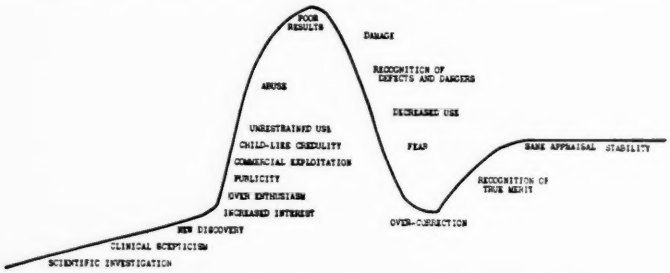

Chart I. The Reaction of the Medical Profession to Anything New. (Modified from the original (1) with the kind permission of Dr. Edwin Davis.)

ciency resulting from vitamin K lack. It is to be remembered however that all prothrombin deficiency is not due to a lack of vitamin K. Various types of hepatocellular damage may cause low prothrombin levels which are not benefited by vitamin K administration. Vitamin K is of no value in hemophilia, thrombocytopenic purpura and allied disorders.

CONCLUDING REMARKS

The importance of nutritional deficiency disorders has become recognized only in recent years. We are now in the period of commercial exploitation and unrestrained use of vitamin products. Although the future will probably see more rather than less nutritional therapy, critical judgment is highly needed at present in the evaluation of etiologic and therapeutic claims.

(I am indebted to Dr. Joseph Berkson of Rochester, Minn., for his assistance in the preparation of the statistical data on erythrocyte counts.)

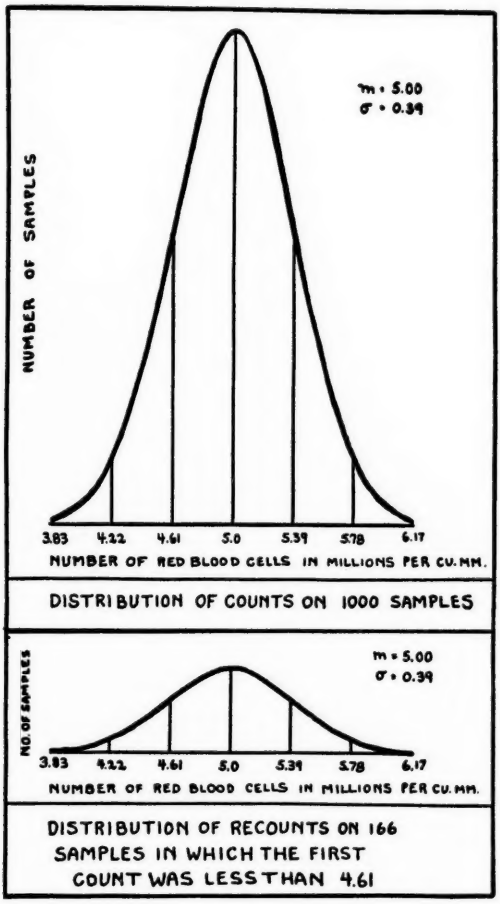

Chart II. Distribution of Red Blood Cell Counts on 1000 samples of a Single Specimen of Blood and Distribution of Recounts on 166 Samples in which the First Count was less than 4.61 million per cu. mm. (Calculated from Data of Berkson, Magath and Hurn (3).)

Chart III. The incidence of Typhoid Fever and the Number of Telephones in Use in Massachusetts 1915–1930. Correlation Coefficient 0.92 P=.01 (Typhoid incidence courtesy of Dr. Roy F. Feemster, Director Division of Communicable Diseases, Mass. Dept. of Public Health. Telephone data courtesy Mr. Donald A. Sawtelle, New England Telephone and Telegraph Company.)

DR. C. E. HERRON—Dr. Strauss has very ably covered the clinical and scientific aspects of the subject "Disorders Due To Nutritional Deficiency". I shall try to avoid repetition as much as possible in my discussion, but due to the newness of the subject in Life Insurance Medicine this will not always be possible. In opening my remarks I should like to emphasize one point which has made itself very apparent in the preparation of this discussion; that in spite of the vast amount of literature on Nutritional Therapy, the wide-spread publicity and use of Vitamin preparation; the field of Nutritional Disorders is still a comparatively new one and a great many of the claims advanced by sincere but overenthusiastic workers must be regarded with critical judgment. Progress has been very encouraging but we have not yet discovered the Panacea. The specific indications for Nutritional Therapy and especially Vitamin Therapy are still definitely limited in scope and we should bear this fact in mind when giving this subject consideration.

With the exception of the Anemias the question of Nutritional Disorders has received practically no attention in Life Insurance Medicine up to the present time. Even the Anemias have not been overly stressed and up until the 1935 code were all placed in the heterogeneous group of 181, Anemia or Chlorosis. The 1935 code was a move in the right direction, in that classifications 354-355-356 were added and should furnish us with some working basis for study in the future.

The importance of the Anemias as an underwriting problem is difficult to evaluate because except in the unusual case we are not favored with the benefit of blood studies. The diagnosis is essentially a laboratory diagnosis and unless blood studies are made almost routinely, the large percentage of applicants for insurance would be passed by because they would not exhibit a degree of Anemia marked enough to be picked up on the usual physical examination. The scarcity of material for statistical study bears this out. In the 1925 Medical Impairment study, with material contributed by com-

panies having 4/5 of the total insurance in force in the United States and Canada there was reported under code 181-

	Exposed to Risk	Actual Deaths	Expected Deaths	Ratio Actual to Expected
Substandard	4,060	24	18.11	133±19

Inasmuch as there were only two deaths in the standard group, the material was combined and the entire group of Standard and Substandard showed actual deaths of 26 or a mortality of 130% ± 17. Deaths from Pulmonary Tuberculosis were 3 1/2 times the normal proportion.

In the 1938 Impairment Study the classifications are broken down to conform to the 1935 code, namely, 354-355-356 and represents a somewhat larger group. It is interesting to note that in a representative group of 50 dead, none was found to belong to the Pernicious Anemia Group. 21 were specified as "Secondary" (356), the remainder were 354–"Anemia not listed elsewhere or no details".

The results of the study were as follows:

	Exposed to Risk	Actual Deaths	Expected Deaths	Ratio
X Substandard	2,071	13	11.27	115±21
B-D Standard	15,553	72	73.78	98± 8

In the Standard section of X there were no deaths and in the Substandard section of B-D there were 9 deaths and a mortality ratio of 106%.

The company ratings averaged 155% for X and 130% for B-D. There were 11 deaths from Pulmonary Tuberculosis against less than 5 expected deaths. Inasmuch as cases of Pernicious Anemia and Anemia due to Tuberculosis are usually declined the ratings for the most part apply to simple Secondary Anemia or "Chlorosis".

Pernicious Anemia presents a somewhat different problem in that, as stated above, Insurance Companies have made practically no attempt to underwrite this type of case or one presenting such a history. The advent of Liver Therapy has, of course, revolutionized the treatment of such cases and has changed the prognosis from a hopeless one to that of at

least a fair expectancy with reasonably adequate treatment. Dr. Strauss has quoted his series of over 500 cases treated by himself and colleagues without a death from Pernicious Anemia. Medical literature contains many such optimistic views and scientific and clinical records to justify them. Drs. Dublin and Lotka in their "Twenty-five years of Health Progress" showed that in a ten year period ending in 1935, there was a decline in mortality from Pernicious Anemia of 32% in men and 53% in women. These figures if brought up to 1940 would in all probability be even more favorable. But Life Insurance Companies in general have not, at least to my knowledge, changed their outlook on this disease appreciably. We are faced with what might properly be classed as a moral hazard in that we have no way of enforcing treatment on those whom we might insure. The problem is very similar to that of the Diabetic. But with the great improvement in treatment and prognosis which has been brought about in persons suffering from Pernicious Anemia in the last ten or fifteen years it would seem that we as medical underwriters could safely begin to liberalize our rules of selection in this group.

The "Disorders Due to Nutritional Deficiency"—specifically those due to Vitamin Deficiencies can best be discussed as a group when applied to Life Insurance Medicine. Our own field of literature and research on the subject is barren—not particularly because of neglect on our part but because the field, except for its fundamental aspects, is relatively new and has received great clinical impetus only in the last few years. It is still too soon to correlate all of the clinical advances with statistical studies such as are best suited to our own field. It is my thought that for the present and perhaps for sometime in the future, the beneficial results of the work in this field will be reflected in our general mortality rather than the results of specific attempts at selection.

When the field is confined to those conditions which we are reasonably sure are the result of Vitamin Deficiencies, the very nature of the diseases tends to make them somewhat

uncommon among applicants for life insurance, at least, in forms severe enough to be recognized. They are after all "Deficiency States" and the deficiency may rest on an economic or physical basis—either has a tendency to weed out applicants for life insurance.

The manifold and complex causes of Vitamin Deficiency are admirably summed up by Gildea (Connecticut State M. J. 4:85 February, 1940) in his recent article.

I The failure to ingest a well balanced diet, i. e., the daily ingestion of meats (including occasional liver or kidneys), milk and eggs, fresh vegetables, fruit and cereals and breads containing some of the grain germ layers. Failure may be due to:

1. Limited economic status.
2. Social customs.
   e. g. Corn and pork diet of South; potato, candy and lean meat in the North; poorly organized vegetation diets, fads such as extreme weight reduction, etc.
3. Ill advised prescriptions of physicians.
4. Personality determined aversion or idiosyncrasies toward food.
   A. The ascetic who looks on food as a manifestation of spiritual weakness.
   B. The hypochondriac who gives up one food after another because of symptoms.
   C. The hysterical person who uses refusal to eat customary food for ulterior but usually unconscious purposes.
5. Practically all illnesses if severe enough may prevent the ingestion of a well balanced diet.

II Disturbed absorption due to Nausea, Vomiting, Chronic Diarrhea of all kinds and lesions producing obstruction.

III Lack of substances in stomach or intestines may prevent utilization of vitamins as for example the lack of intrinsic factor in Pernicious Anemia.

IV Individual requirements may be high.

V Constitutionally people differ markedly in the amount of Vitamins necessary to prevent symptoms.

VI All conditions which increase metabolism increase vitamin requirements. Growth, Pregnancy, Fever, Hyperthyroidism and common examples of conditions which elevate food and vitamin requirements.

VII That the ingestion of certain toxic substances may increase vitamin requirements has been demonstrated recently.

Dr. Strauss has pointed out the role of the various vitamins as we now know them.

To summarize briefly—

Vitamin A and its role in Xerophthalmia and night blindness.

The more complex Vitamin B and its components and their roles in Pellagra, Beri Beri, the perepherial neuritedes and cardiovascular failure.

Vitamin C and its important role in Scurvy.

Vitamin D and its specific benefits in Infantile Rickets, Spasmophilia and Osteomalacia.

Vitamin E is still in the experimental state and its application to humans is not yet established.

Vitamin K shows great promise in specific hemorrhagic disorders.

In closing my remarks I would like to mention a few of the points which should be of especial interest to us as Medical Underwriters.

1. The finding or the history of Anemia, while important in itself should also put us on guard to investigate for a possible underlying cause which may be of more serious character than the Anemia. The fact that the degree of Anemia may be slight does not necessarily mean that the cause is of benign character. Special emphasis should be placed on history—including family history, the finding or history of Achlorhydria. Statements from attending physicians may be helpful.

2. As we all realize the marked degrees of Avitaminosis necessary to bring out the clear cut clinical pictures discussed and illustrated by Dr. Strauss do not present themselves for insurance and as yet do not comprise a great underwriting problem.

3. There is, however, a large number of cases of subclinical Avitaminosis which we must be on the alert to detect. At the present time this is extremely difficult because of our rather meager knowledge on the subject as a whole. The many possibilities held out by Vitamin B seem to be of especial interest, and we hope that the constant research which is being carried on in this subject will soon give us some of the answers to this complex Vitamin.

PRESIDENT FROST—I think perhaps you will all agree with me that Dr. Strauss has cleared the air considerably.

I was interested to note the other day, Dr. Strauss, a reported discussion by Dr. Castle; I think it was before the American X-ray Society, and it was with reference to leukemia and its treatment by means of Vitamins.

I would be interested if you would give us a few words as to that.

Are there any other questions any one would like to ask Dr. Strauss?

DR. STEELE—I want to take this opportunity of thanking you, first, for such a marvellous program, and for the change from dull statistics to clinical medicine.

I want to thank Dr. Strauss for such a wonderful paper. You made two statements, Doctor, which I should like to repeat. First, you stated that it was estimated that in 1938 a hundred millions of dollars were being expended annually in the United States for Vitamins. The Lord, Himself, only knows what it is today. And, by the way, I don't know whether you noticed the paper today or not, but it said the Lapans were using lipsticks with Vitamins in them, so as to strengthen their boy friends.

You also stated, in closing, that deficiency diets have become recognized only in recent years, and that we are now in the period of commercial exploitation and the unrestrained use of Vitamin products. You also said that critical judgment is highly needed at the present time in the evaluation of therapeutic claims.

No truer statement was ever made. I know that must be true when my wife goes downtown and pays $1.50 for some Vitamins to feed the dog with.

Personally, I was most interested in your remarks concerning Pellagra. I am a Southerner and I was what is called a Health Officer for seven years in a southern city. During the seven years, no one ever heard of nicotinic acid. Yes, and Pellagra is consistent only by its inconsistencies.

You showed a particularly fine picture during your talk. You know, they always say if you have a dull spot here, you have one there. You had one on the right arm and another one on the left. Well, that is a true statement, and about the only one on Pellagra.

I am also of the opinion that the average medical director knows so little about Pellagra that he has no right to attempt to rate it. I make this statement after looking over the rating charts of several of my friends.

In closing, I just want to bring this point out, which I got from Dr. Harris of Framingham. Liver and liver extracts

cure people. Secondly, liver is a depositary for all the Vitamins, and it is known that nicotinic acid is present in the normal liver.

I should like to know whether you agree with those statements or not.

PRESIDENT FROST—Gentlemen, this paper is open for further discussion.

* * * * * *

DR. ALLISON—I certainly want to take this opportunity to thank Dr. Strauss for leading me a little way out of my confusion. Like most medical directors, I have sort of stood on the sidelines of the advances in biochemistry, so that when they talk about what happens in the chemic gizzard and elsewhere, I confess a great deal of ignorance.

I agree with Dr. Strauss that we are in the stage of abuse, and as to the fact that so many millions of dollars are being spent, I don't know just how to account for it, but somebody must have let the word get out. I suppose you could correlate it to the alcoholic consumption in this country. I understand that in that same year, 1938, there were over one hundred million gallons of hard liquor consumed in this country. So I don't know; maybe there is some correlation there.

From the Chair of a medical director, I think that few of us see the Vitamin deficiencies. A lot of them are subclinical and certainly in the sections of the southwest, where I come from, you don't see much Pellagra.

However, there is a very enthusiastic use of some of the newer Vitamin preparations, especially B_1. I don't think any one there regards it as a cure-all, but I do think that a great deal of good is being accomplished with its use. I know that some of my friends there feel that their fallacious reasoning may be justified when the person with dyspnoea, with numbness and tingling and those various symptoms that are so hard to evaluate and not very specific of anything, comes to

them and gets relief on Vitamin B_1 therapy. And so they have gained a great deal of confidence in its use. They use it in individuals who have hyperthyroidism. And I think it is being used routinely in some of the hospitals, following abdominal surgery. I think there is a good indication for its use there.

I don't think there is a great deal of enthusiasm in connection with some of the neuritides, although I have had a personal experience that was rather hard to explain. An elderly gentleman was sent to me by his nose and throat man; he had buzzing in his ear, and he wanted to find out if there were any organic diseases present. Well, I couldn't find any. So I sent him back to the other man, but he sent him back to me. So I gave him some Vitamin B_1, and about four days after treatment, he was all right, and he came around to see me and wanted to know where he could buy the thing in wholesale quantities. I guess he wanted to give it out to some of his friends.

I think that the life insurance medical director certainly is going to have to be very reluctant to accept the claims that are coming from some neurologists and psychiatrists. It may be that what they have to say is true, and that a definite relationship does exist between the nutritional deficiency and some of the more seriously regarded mental diseases. I think that time will prove that.

But I think that we might make some apology and some plea for just a little fallacious reasoning in continuing the use of Vitamin B_1 especially, and certainly I think the use of Vitamin K is going to grow daily.

* * * * * *

Dr. Old—I attended my first meeting of the A. M. A. in Atlantic City. At that time, one of the common causes of the large, white kidney was supposed to be due to alcohol. A young man got up and read a paper before that Association,

in which he stated that he had gone to an institute where there were nothing but chronic alcoholics, and he made autopsies on alcoholics who had died in that institution. He didn't find among that group any large, white kidneys.

That man was Dr. Richard Cabot of Boston.

In other words, he said that was a myth, which he disproved.

I now come to Boston in a year of extensive exposition of this present craze throughout the country of Vitamin deficiency and treatment, and it is a fad. In fact, you don't have to have much imagination to think, in the near future, of some woman who is up-to-date, and especially if she is acquainted with commercial houses that produce these things, giving a luncheon, and on the invitation, asking: What is your chief complaint? And upon the complaints, she will have groups arranged. Conversation will be something like this. "Well, you know, at dusk, I can't see what my neighbors across the street are doing as well as I used to." All right. Vitamin A goes in for her. And another one will say that she has a poor appetite, and she has some pains in her joints, or something like that. So she will get Vitamin B. And another one will say that sometimes she suffers with bleeding gums. Well, there goes your Vitamin C. Still another will say she is quite bow-legged. There will go your Vitamin D. And another one will say that she has always wanted to have children and never had any children. And there goes your Vitamin E.

Now, that is a perfectly plausible thing to occur.

But, from the insurance medical standpoint, I think that the important group that Dr. Strauss spoke about is the Pernicious Anemia group. Pernicious Anemia occurs at the age period between forty and sixty-five years of age, which is exactly the same period where we get our cancer cases.

I should like to ask Dr. Strauss this question. How can such be differentiated? That is, how can these Pernicious Anemia cases be differentiated from those with cancer of the stomach? Also, there is another common group that we get now, of myxedema. Another point is this. How does the

case of Pernicious Anemia, the case of cancer of the stomach, and the case of some of these anemias, respond to histamine or to a weak alcoholic solution, so that there would be a possibility of differentiating one from the other?

PRESIDENT FROST—I shall now ask Dr. Strauss to close his paper.

DR. MAURICE B. STRAUSS—I did not want to leave you with the impression that I decry the use of Vitamin B_1 in conditions for which there is an indication. In fact, it is only eight years ago, in this vicinity, that I was called a murderer by a prominent physician, because I insisted upon treating a woman with polyneuritis of pregnancy with crude Vitamin B_1 preparations instead of having a therapeutic abortion. Only a year after that, it was considered quite silly for us to begin to treat alcoholic polyneuritis with B_1, rather than simply by stopping their alcohol.

Vitamin B_1 has a distinct role and it is definitely indicated in certain conditions.

Dr. Frost inquired about the recent report of Dr. Castle in his address before the American Roentgenological Society. Dr. Castle was very much chagrined by the unfortunate and incorrect newspaper report. In fact, I know that he requested the newspapers to publish a retraction, and, as far as I know, none of them considered that news.

Achlorhydria exists in twenty per cent of adults at about age forty to sixty. It is in no sense pathognomonic with cancer of the stomach and Pernicious Anemia.

From an insurance standpoint, the best way to be sure that a Pernicious Anemia doesn't have cancer of the stomach—and one per cent of the patients do—is to defer insurance until one or two years have elapsed after the diagnosis of Pernicious Anemia has been made, or else insist on a G. I. X-ray examination.

As far as insurability is concerned, I should be very hesitant about wishing to personally insure the life of any one who

gave a history of either Polyneuritis or Pellagra. When one has either of those conditions, it suggests poverty, bad diet, gastro-intestinal tract disturbance, hyperthyroidism or other serious organic disease as an underlying mechanism.

Nicotinic acid is certainly present, as are most of the vitamins, in liver, and liver extract is a very satisfactory way of administering many of the components of the B complex.

I think that probably I should close with reminding you that there is a very satisfactory way of getting an adequate supply of all the needed vitamins. They are marketed by three very common types of stores; the grocer, the butcher and the dairyman.

There is no better way to get an adequate intake of not only all the known vitamins, but all the unknown ones, than from a good, well-balanced meal, and I think it is, indeed, appropriate that Dr. Frost suggested we do that next.

PRESIDENT FROST—Dr. Strauss, we thank you for your excellent paper.

PRESIDENT FROST—One of the problems in which I have always been interested, as a medical director, is that of allergy, with particular reference to asthma.

I have asked Dr. Francis M. Rackemann to come here to discuss this subject. Dr. Rackemann and I have known each other for a good many years. As a matter of fact, he was a year ahead of me in medical school and hospital service all through the different branches. I know that he became interested in allergy in the early stages of the development of the knowledge with respect to it.

I don't know that I need to tell you that his name is a byword in circles where allergy is discussed.

Dr. Rackemann was graduated from Harvard Medical School in 1912. He is now Lecturer in Medicine at the Harvard Medical School.

Dr. Rackemann!

THE TWO KINDS OF ASTHMA

By Francis M. Rackemann, M. D.
Lecturer in Medicine, Harvard Medical School
Physician, Massachusetts General Hospital

Asthma is not a disease; it is a symptom. One must recognize the various types and kinds of asthma and the various causes of asthma before one can understand the case at hand to advise the proper treatment and to estimate the probable outcome.

There are two principal kinds of asthma. There is the kind that depends upon allergy and in which the attacks of asthma bear a definite relation to exposure to foreign substances; to different seasons of the year or perhaps to some special food. It is called "extrinsic". The other type of asthma depends upon changes inside the patient. It is called "intrinsic". Whatever is the cause, it is something that the patient carries with him wherever he goes.

It is the first type, extrinsic asthma, which has given this subject its popular appeal. When the cause of trouble is known, a good deal can be done about it. Here is Peter, for example. He is an expert in making salads of all sorts, and lobster salad in particular. But, every time he handles the lobster meat, his fingers will begin to itch, and pretty soon he will begin to sneeze and wheeze and then he has to go home.

The symptoms depend upon his capacity to develop a sensitiveness to foreign substances. As a result of his occupational exposure to the lobster meat, he has become sensitive to it.

A skin test to lobster was made by taking some of the meat and grinding it with salt solution and putting a drop of the extract on Peter's arm and making a light scratch through it. Even if the crude extract is diluted ten or one hundred times, it still shows a positive reaction. Peter is highly and specifically sensitive to the lobster meat; he provides a text book picture of this disease.

Another patient is a plant pathologist, whose business it is to go around to various commercial greenhouses and tell the proprietor what is wrong with his tomato plants, when the leaves turn brown and the fruit does not develop. That plant disease is due to a fungus called Cladosporium Fulvum and the man has become specifically sensitive to the spores of the fungus.

He shows markedly positive skin tests when the spore extract is applied to a scratch in his arm. It is important to observe that if this man stays away from the greenhouse, he is perfectly well, and remains just as healthy as we are.

The bakers who have become sensitive to wheat present a much more common and more practical problem. The diagnosis is easy because the asthma is directly related to contact with wheat in the occupation. When the baker is working, he is in trouble, but when he is away, he is all right.

Obviously, it is the clinical history which is the important factor in the diagnosis of all these cases. From our study of hay fever, we have learned that in taking the history, the dates are essential. With dates, one can make a good diagnosis, whereas if one persists in saying "Seven months ago" one is likely to miss the point.

One time we had an earnest student who was taught to record time as—so many months ago. He went out of his way to say that something happened five months ago, and that something else happened seventeen months ago. The difference between five and seventeen was twelve. The student knew that the trouble developed each year around Labor Day. But he took pains to throw away that important information. If he had written the dates, it would have been obvious that the trouble which occurred each year at Labor Day was due to ragweed.

Extrinsic asthma, and hay fever, too, are diseases which begin in early childhood, and that suggests another point in the history. It is important to write down not only the date of onset but also the age at onset. If the disease began at age twenty, it is quite different from another kind of asthma

which did not begin until the patient was aged fifty. The history is the essential method of making a diagnosis. One must find the age at onset and one must trace the patient's story from that age down to the present time, noting the dates of each new attack and the dates of any changes in environment or occupation as well as the dates of intercurrent diseases or of operations. One must try to account for all of that time. If there was a period of freedom for several years, one must find the reason. Perhaps the patient married and moved to the new house and so escaped from the dust at home. Perhaps it was the time when new furniture, especially new bedding, was obtained. Perhaps the attack ended because she went to the clean environment of the hospital.

Meantime, one must know what are some of the possibilities that might account for asthma. Cats and dogs are obvious. Pollens are obvious. But it is only in the last five or six years that we have taken interest in the molds that are in the air. Like pollen, the molds, too, reach a peak in September and October. Molds must be considered as possible causes, when treatment with pollen is not satisfactory.

Molds cause trouble in another way. Last September, I saw a pathetic girl of twenty, who had had asthma all her life. She had been through high school, and had just started her first job. She came to the clinic, just about "all in." She could just barely breathe. We put her in the dust free room at the Massachusetts General Hospital and in forty-eight hours she was greatly improved, and in four days, most of her asthma had disappeared. Her skin tests were negative except for a slight reaction to feathers.

The striking change proved that her trouble depended upon some dust at home. If we had sent her home, she would have had asthma again the first night, and so it was arranged that she go to a boarding house, and there she did well. In the meantime, the social worker and I went to the home, up two flights of stairs, and to the rear. The place was dirty, and the furniture broken down. The girl had been sleeping in the front room on a day bed stuffed

with kapok or "silk floss" as it is called. Incidentally, little brother Johnny, age twelve, had asthma, and the father had touches of asthma when he had a cold. The back room, however, was sunny and fair sized, and the mother told us that the landlord had been talking about putting new paint and wall paper on the back room. The social worker approached the landlord. The back room was painted and the wall paper changed. New mattresses were obtained and, after about a week, the girl went home. Her asthma did not return and in the three months since then, she has had no trouble and is back on her job.

That is the sort of thing that can be done for these cases. The prognosis is important. If this girl can always live in a clean environment, there is no reason why she will not live to a ripe old age.

Kapok (silk floss) has caused much trouble. When it is new, the fibres are clean and firm; but when it is old, the fibres break down to a fine dust. The reason why kapok breaks down is because molds grow on the kapok fibre.

Cotton seed may also be of importance, but we are not sure about it yet. What I like, however, is to have patients sleep on hair mattresses and pillows, and the new sponge-rubber mattresses and pillows give great promise.

Skin tests have given this subject its glamour. Their importance, however, is not in making the diagnosis so much as in confirming the diagnosis which the history suggests.

Skin tests are in no way fallible. One finds positive tests in normal persons when there is no clinical sensitiveness to go with them: "False positives," one might say. The skin test will often persist after symptoms go and that, we think, is an explanation for some of the false positives. The skin test is a record of previous experience.

Skin tests may change from time to time, and this case is interesting because it shows a little about the natural development of allergy in young persons. Here is a child I saw at the age of seventeen months. At that time, he had a good

reaction to egg albumin, and a little reaction to orris. We took him off eggs; his eczema was much better and his asthma was under good control. Eleven years later, at the age of twelve, he was tested again. This time, the egg test was negative, but the ragweed test had become positive and other dusts reacted. As a baby he was sensitive to egg, but now as a boy he is sensitive to dust. The picture has changed from foods in children to dust in adults.

A man who presented himself in his first attack of hay fever in the early spring had negative skin test, but two years later, his tests were markedly positive. Evidently his sensitiveness had developed in his nose first; in the skin later.

In contrast, here is a woman whose tests become smaller from year to year. She has had no trouble since 1933. Meantime, the amount of treatment she has to have is markedly reduced so that, now, four or five doses of pollen extract are all that are needed to maintain her ground. Skin tests, then, may be positive and mean nothing, or they may be negative even when the patient is clinically sensitive.

Intrinsic asthma is the other kind of asthma. It is a different disease and it presents a much more difficult problem.

In a group of 283 cases, representing perhaps twenty per cent of all patients with asthma, it has not been possible to demonstrate any clear relation between the asthma and any change in environment or occupation. In a bad attack, these patients come to the hospital and improve, perhaps, but they do not show the same striking sudden improvement as occurs in the extrinsic group. Whatever is the cause of the trouble, it is something which they carry with them. They have a fundamental defect, which results in their asthma.

The histories of these cases provide a basis for dividing them into groups.

The first group is small. It includes a few patients who started off perhaps in childhood with a typical story of extrinsic asthma having a clear relation to changes in their environment. Later, and often quite abruptly, the attacks be-

came more numerous and the intervals between them shorter until finally the asthma has become essentially continuous.

In another group, there was a similar story of extrinsic asthma at first, and then came a free interval lasting for a number of years. Then, at the age of about fifty, asthma came again quite suddenly, and this time bearing no relation to changes in environment or season. The early story of extrinsic asthma suggests perhaps that the new attack depends upon a conditioned reflex. When something else goes wrong the old condition is reawakened, and the asthma recurs.

In a third group, the asthma depends upon colds. "Asthmatic bronchitis" is a good designation. The group is large. Why do these people wheeze with their colds? Sometimes, one can show that they are slightly sensitive to some dust factor, since after the removal of the cat or the pillow, the colds may recur but this time without a wheeze; but in other cases, there is no such easy answer.

The fourth group is most important. It consists mostly of men who have been healthy all their lives until, at about the age of fifty, they begin quite suddenly to have asthma. What does that mean? There is no evidence of allergy in the cases, and no evidence of previous asthma. The asthma starts without obvious cause, and when it starts, it is persistent and goes on from bad to worse. One talks about "primary idiopathic emphysema," of tuberculous glands at the hilus which become active; or of "sinus bronchitis," but it is all indefinite. Positive skin tests are observed in a few of the cases, but there is no evidence that the corresponding foods or dusts have any causal relation to the origin or the course of the asthma. Some of the cases have a positive family history of allergic disease. This group constitutes an important problem.

Finally, there is a very special group in which asthma is associated with a severe chronic vasomotor rhinitis. The prognosis in this group is serious; out of fifty cases, ten have died and that is a mortality of twenty per cent. The majority of the cases are in women, and the age of onset is young; only thirty-seven years. The typical story is of chronic nasal

trouble which has existed for some years and then, for the first time, asthma comes with it, and almost from the start this asthma is of maximum severity, and, as I say, twenty per cent have died, and died of asthma. The duration of the disease is, on the whole, short; averaging about four years. A good many, if not all, of these cases have disturbances in their sinuses. Many of them have had previous operations and three of them were operated in our clinic but all without success.

Complications in the nasal sinuses represent a problem that is common and difficult. I see a certain number of people who never did have asthma to any extent until the nose was operated upon and then, in place of having a relatively mild disease, they have been in real trouble. And so, I have seen operations on the nose and throat do harm, I think, about as often as I have seen them do good. On the other hand, I have also seen asthmatic patients who have had their nasal sinuses operated and the asthma disappeared.

Results in asthma are always hard to evalute. The patient may move to a different home; he may move into the front room from the back room. Perhaps his cat was run over. It is often hard to be quite sure that extrinsic factors really are excluded when one would like to think that the good result depended upon the operation.

I believe that the lesion in the sinuses is part and parcel of the lesion in the bronchi; that the sinus disease is a part of the asthma, and certainly not a cause of the asthma. It goes with the picture.

The pathology of the lungs in asthma is always striking. When the lungs are removed, they do not collapse; they make a perfect cast of the chest wall.

On cross section, one can see plugs protruding from each of the bronchi, both large and small. These plugs are made of dried mucus and since they shut off the air, they produce death by suffocation. The heart remains sound. In several cases it was found beating long after respiration had ceased.

Under the microscope, the picture is striking. In the first place, there is a tremendous emphysema. There is a coalescence of alveoli. There may be a few small areas of infiltration here and there, but as Dr. Tracy B. Mallory has pointed out to me, the important lesion is the increase in the size and in the number of the mucous glands, which are in the walls of the bronchi.

The mucosa is folded, the muscles are enlarged, perhaps, but the mucous glands are large and prominent. Meantime the rest of the lung is clear. There is very little in the way of any peri-bronchial inflammation. There is no evidence of an infectious process. The trouble seems to be a physiological disturbance largely concerned with the bronchial glands, and the theory comes that the plugs form because of the overactivity of these bronchial glands.

When one instills lipiodol down the trachea, the X-ray shows that it comes to a stop in the bronchial lumen, and one can see that there is a cup shape at the end of the shadow, as though the lipiodol fitted over the bronchial plug. The plugs are there in life.

Do all of the fatal cases have this same plug formation? I cannot answer that. In the original paper by Huber and Koessler, published in 1922, similar plugs are mentioned, but their practical importance is not stressed. At the Massachusetts General Hospital, we have observed a considerable series of patients who died of asthma—not merely with a history of asthma—and it becomes very important to study the pathology carefully to see whether there may be some other mechanism besides plugs to cause their death.

In conclusion, may I emphasize the fact that in the extrinsic group of cases, where allergy is important, and the asthma is due to foods and dust, and occurs in isolated attacks with free periods between, and where one can show by the history that these attacks are related to some definite change in season, occupation, environment, bedding, clothing, animals or perhaps some other special circumstance, I believe the prognosis is good. In the right environment, these patients

do well. The troublesome cases begin to have asthma later in life, and their asthma, once begun, becomes persistent and continuous, regardless of changes in season or environment or occupation. These are the bad cases. To determine the nature of their trouble will require much further study.

PRESIDENT FROST—Dr. Henry B. Kirkland, Assistant Medical Director of the Prudential Insurance Company of America, has kindly offered to discuss this paper, perhaps from another angle, as Dr. Rackemann has indicated.

Without further ado, I will ask Dr. Kirkland to come forward and give us his discussion of Dr. Rackemann's paper.

Dr. Kirkland!

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DR. KIRKLAND—Dr. Rackemann has given us a very lucid analysis of the classification of asthmatics. It is evident that painstaking study of the individual case is essential in such classification, and it is on this account that the underwriting of risks giving a history of asthma is fraught with so much uncertainty. We are unable in most of our insurance selection work to analyze allergic backgrounds adequately, and are compelled for the most part to evaluate insurability on a very arbitrary basis.

Our statistics are unfortunately of little practical assistance to us in this connection. In any large series we are confronted with erroneous reports of causes of death, and it is quite impossible to attempt to correct relatively crude data emanating from histories which are fragmentary at best. Our errors of judgment may lead us far afield in two directions. We may take adverse action in cases in which there is a long duration of symptoms from the onset, although there is abundant evidence that this factor is not inconsistent with almost the maximum period of life. On the other hand, we may pass lightly over a history of two or three apparently negligible asthmatic attacks in cases which, on more searching

analysis, would show other findings warranting inclusion in the more unfavorable group.

Although errors will be readily made in evaluating risks with histories of asthma in the younger age groups, we must most certainly be extremely conservative in our judgment when we are dealing with individuals further on in life in whom cardiovascular changes of the degenerative type may have already had their inception. The differential characteristics of allergic asthma and of the so-called cardiac asthma, the latter being not the happiest of descriptive terms, are of course well known to you. Certain points will bear reiteration more for the purpose of clarifying our conceptions of the whole problem than as a basis for selection, since in most instances concomitant unfavorable features will be decisive.

Some forms of left ventricular failure produce a paroxysmal dyspnea undistinguishable by symptoms, signs, or therapeutic response from bronchial asthma. Although it has been said that wheezing respiration is in almost all instances pathognomonic of an allergic background, this viewpoint does not seem entirely tenable in view of the fact that this sign is dependent on the same factors as those concerned in allergic asthma, that is, narrowing of the lumina of the smaller bronchioles, whether this be by direct muscular spasm, swelling of the bronchiolar mucosa, or edema fluid, mucus, or other detritus producing obstruction. Where the asthmatoid type of respiration dominates the picture, real difficulty is encountered in establishing a diagnosis. Even eosinophilia, response to adrenalin, and venous pressure determinations may be equivocal in certain cases. Circulation time estimations may eventually be more helpful when criteria of normal have been more rigidly established. It would appear that the total circulation time in allergic asthma is normal, except during the paroxysm, whereas in most left sided failures total time is prolonged, the pulmonary time constantly and the arm-to-lung time frequently.

It has been pointed out that a not inconsiderable group of individuals develop asthma for the first time in the sixth

and seventh decades of life. The mechanism is obscure, and it cannot be said with any surety that arteriosclerosis is a factor, but it cannot be doubted that most cases of this type present insurmountable diagnostic problems, and from an insurance selection standpoint should be considered unfavorable even though the attacks are mild and widely spaced. I am not familiar with the exact underwriting practices of more than a few companies in regard to asthma, but I have the distinct impression that the age of onset of this condition is not always accorded the attention that it seems to deserve. It is probable that it would be sound practice to subject all asthmatics above the age of 45 or 50 to exceedingly searching scrutiny and to refuse to consider on any basis those who have their first attacks at about this age. The establishment of a satisfactory definite line of demarcation would depend on a more detailed study than I have been able to make.

A few words regarding the cardiovascular aspects of asthma may not be amiss, and in this connection mention might be made of the roles played by emphysema and pulmonary fibrosis in the causation of right sided cardiac involvement. As has been pointed out, it is thought by most observers that heart disease is rare in asthma, and this conclusion is apparently based on a fairly large number of reported necropsies. There is, however, by no means complete unanimity on this point, and even so acknowledged an authority on general pathology as Dr. Paul Klemperer, with whom I was discussing this subject only the other day, believes that some degree of right sided involvement may occur relatively early, as duration is measured in so chronic a condition as asthma. We took occasion to review a number of the more recent Mt. Sinai protocols of cases in which the presence of asthma had been unequivocally established clinically, and found that in every instance some degree of hypertrophy of the myocardium on the right side had been described. Some of these individuals were quite young. It seemed of significance to me that in no one of these cases was pulmonary fibrosis of appreciable extent present, although emphysema of some degree, at times

very advanced, was a constant finding.

Here again we find an intangible factor, so many of which seem to be encountered in the study of asthma and its complications and sequellae. The very usage of pathological descriptive terms demonstrates how greatly the designation of present or absent myocardial hypertrophy or dilatation depends on the judgment of the individual observer, who is, more likely than not, primarily interested in other aspects of the case with which he is engaged. The same question can be raised in regard to changes in the pulmonary arterial tree. In fact, it would seem that there would continue to be doubt as to the exact amount of cardiac involvement secondary to asthma until more accurate post-mortem studies of the exact relative weights of the two ventricles are made or until some clinical method is devised to yield more definite information on this point. There would seem to be promise in the procedure developed by a member of this Association, Dr. George P. Robb, and his associates for the visualization of the heart chambers and great vessels by diodrast roentgenograms, although this will of course depend on the fixing of definite standards of normalcy.

Regardless of the doubtful pathological aspects, the fact remains that from a clinical standpoint uncomplicated asthma does not seem to affect longevity seriously. An interesting theory advanced to account for the sparing of the heart is that during each asthmatic paroxysm there is diminished cardiac filling due to a drop in the negative intra-thoracic pressure. Some observers have established that during the attack the heart often appears radioscopically smaller than normal. The behavior of the blood pressure during paroxysms is controversial. Many cases show a tendency to or actual right axis deviation in the electrocardiogram, but rarely any evidence of myocardial change. Even the former may be due to the occurrence of a low diaphragm in asthma and moderate degrees of emphysema, with consequent change in heart position, rather than to right sided cardiac hypertrophy. Although these various factors may be subject to variation in interpretation, there

can be no question of the fact that right sided heart failure, with death, is an uncommon occurrence in asthma unless the condition has progressed to a stage of more than moderate emphysema or of actual extensive pulmonary fibrosis.

It may be of interest in this connection to touch briefly on the actual occurrence of cardiac involvement and failure in the presence of extreme emphysema and advanced fibrosis, especially in pneumoconiosis. In these conditions there is a very marked shrinkage of the pulmonary capillary bed associated with mechanical obstruction. It is improbable that the nodulation found in certain cases of silicosis has any great influence on the capillary flow, since the most extreme dyspnea may be encountered in those cases in which the lesions have a cloudy or hazy appearance by X-ray often not even considered diagnostic of the condition any more than the Roentgen appearance in asthma is characteristic. There is, however, reduced distensibility of the lungs, and dyspnea is constant even at rest due to early response of the vagal endings in the pulmonary tissue, with rapid shallow breathing, and the unusual effort required of the intercostal and accessory respiratory muscles. These conditions are not spasmodic, as are the paroxysms of asthma, and a constant strain is thrown on the right side of the heart and the pulmonary circulation. Enlargement of the right ventricle and dilatation of the pulmonary artery occur; the individual muscle cells of the former hypertrophy, and failure ensues with terminal dilatation.

In our cardiac clinic at Bellevue Hospital we are often called upon to render opinions concerning the cardiovascular status of asthmatics and of patients suffering from other chronic pulmonary conditions. Surprisingly rarely are we able to identify definite heart involvement. The signs and symptoms at first suggesting early failure are, after investigation, ascribed to the underlying pulmonary disease. Only in those patients in the age groups in which complicating sclerotic disease is present are there definite signs of cardiac failure, and in these instances, although it is usually impossible to separate the two etiological components completely, the evidence almost always

seems to point in the direction of the generalized arteriosclerosis.

I sincerely feel that little could have been added by this discussion to the very comprehensive survey of the asthma problem which Dr. Rackemann has given us. I shall be happy if my remarks have in some measure supplemented his presentation along lines on which the scope of his thesis did not permit him to touch.

PRESIDENT FROST—We will devote a few moments to the discussion of this problem, from the life insurance point of view, or otherwise.

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DR. BARTLETT—It is quite evident from what Dr. Rackemann said, that we would not be interested in taking intrinsic asthma for insurance, because the mortality would be considerably higher than we are interested in. However, we do, occasionally, take extrinsic asthma, and I wonder if Dr. Rackemann could tell us any method by which we could determine the factors in connection with this.

PRESIDENT FROST—Are there any other questions or are there any other comments?

If not, I am going to ask Dr. Rackemann if he would care to close the discussion.

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DR. RACKEMANN—I am personally grateful to Dr. Kirkland, for he certainly has amplified what I had to say, and it fitted in exactly right. I could add one little paragraph to what he said. He touched on pneumonokoniosis and silicosis. He might also have added that asthma, as a symptom—and let me emphasize that, as a symptom—may be due to other things. I have seen a gumma of the larynx cause asthma. Foreign

bodies in the trachea in young children can cause it. Carcinoma of the bronchus can produce asthma.

The other day, we had an interesting case of a colored girl, with tremendously big glands, which caused typical asthma, unrelieved by adrenalin. X-ray reduced the size of these glands, and relieved her entirely.

Now, for the rough and ready diagnosis of extrinsic asthma. I think that Dr. Kirkland is quite right that the age at onset is, on the whole, very important. The presence of asthma in the family is also very important. The inheritance of the capacity to develop sensitiveness disappears in point. The occurrence in other manifestations of allergy, aside from asthma, is important, such as the child has or did have eczema and eczema of a special type, such as distributed to the face, neck, elbows and behind the knees. When you see that, that is evidence of allergy.

Also, the appearance or the occurrence of good idiosyncrasies, along with the asthma, is important. I have in mind the child who has asthma from feather pillows. Multiple sensitiveness is the rule, and the capacity to develop this sensitiveness advances to a considerable degree.

Those are the only points I wish to emphasize. Of course, there is, primarily, the history, and you have to merely amplify and check that history.

PRESIDENT FROST—We are very grateful to you, Dr. Rackemann, for bringing us this message.

Startling developments during the last four or five years in the way of chemotherapy, of course, are news to none of us. From the life insurance medical standpoint of view, we cannot but wonder just what the future of this type of therapy holds for us, and what effect it is going to have upon our mortality, for instance, as compared to pneumonia and other types of disease.

This knowledge is advancing, also, by leaps and bounds.

We have been fortunate enough to persuade Dr. Chester S. Keefer, the Wade Professor of Medicine at the Boston University School of Medicine, to come to us this afternoon and give

us "The Present Status of Chemotherapy in the Treatment of Infectious Diseases."

I can assure you that Dr. Keefer has no superior in this department of his knowledge upon this subject.

Dr. Keefer!

THE PRESENT STATUS OF CHEMOTHERAPY IN THE TREATMENT OF INFECTIONS

By Chester S. Keefer, M. D.

Wade Professor of Medicine, Boston University School of Medicine

(From the Evans Memorial, Massachusetts Memorial Hospitals and the Department of Medicine, Boston University School of Medicine, Boston, Massachusetts.)

It is, indeed, a great pleasure to be invited to take part in your program this afternoon, and I propose summarizing some of my observations on the use of various chemotherapeutic agents in the treatment of various infections.

During the past five years, we have all witnessed great changes in the treatment of infectious diseases. These advances have been due, in a large part, to the introduction of three drugs. They are: (1) Sulfanilamide, (2) sulfapyridine, and (3) sulfathiazole.

Before discussing their relative merits, I shall make a few remarks concerning their characteristics. Sulfanilamide is much more soluble in water than either of the other two. It can be dissolved in 0.8 per cent solution, and, when it cannot be given by mouth, it can be given subcutaneously.

Sulfapyridine and sulfathiazole are extremely insoluble in water. The sodium salts of these two drugs are soluble in high concentration and, when necessary, they can be given intravenously. They cannot be given subcutaneously with safety since they are alkaline, having a pH between 10.4 and 11.

All three of these drugs are absorbed readily from the gastro-intestinal tract and diffuse rapidly through the various tissues of the body. There is some evidence that sulfathiazole diffuses into the sub-arachnoid space less readily than sulfanilamide or sulfapyridine. It is, therefore, advisable not to use sulfathiazole in the treatment of any case of meningitis unless the level of the drug in the spinal fluid is carefully controlled.

Once these drugs enter the blood stream, they are soon rendered inactive by means of acetylization, and a much more insoluble product is formed. The acetylated fraction is not only inactive, but it may precipitate out in such tissues as the kidney and produce blockage of the renal tubules, and hematuria. In some instances, oliguria and renal insufficiency may follow. All three drugs are toxic to some degree but, by and large, the three most important toxic manifestations of these drugs are acute hemolytic anemia, agranulocytosis and, in the case of sulfapyridine and thiazol, renal insufficiency. In our experience, we have never observed renal insufficiency following the use of sulfanilamide. Sulfanilamide has been used, of course, much more widely than either of the other two drugs. Indeed, I was informed about six months ago that approximately 800,000 pounds of sulfanilamide had been consumed by the American public during the last five years.

Sulfanilamide: This drug has had its widest use in the treatment of hemolytic streptococcal, gonococcal, meningococcal, and urinary tract infections.

In the case of hemolytic streptococcal infections, the mortality rate is reduced in patients with bacteremia, meningitis and in puerperal sepsis. It will also shorten the duration of the disease in erysipelas and hemolytic streptococcal pneumonia.

In the case of hemolytic streptococcal empyema, there is some evidence that it will reduce the fatality rate, although there is very little evidence that it causes a very great reduction in either the frequency or the duration of the disease.

In the case of meningococcal infections, the fatality rates have been reduced to about 10 per cent. This is a remarkable reduction over what has been possible in the past in the use of other agents, such as specific serum.

In gonococcal infections, we know that in many instances of local gonococcal infections of the genito-urinary tract and of the conjunctivae, the local focus may be sterilized within a few days. There are, to be sure, failures in the treatment of gonococcal infections with sulfanilamide, but one can say with a certain degree of assurance that this group of drugs is more effective in controlling these infections than any that have been introduced previously.

One may observe a sterilization of infected synovial fluid within a period of forty-eight hours, and the same is true in many cases of localized gonococcal infection elsewhere.

In the urinary tract infections, one finds that the best results have been obtained by the use of sulfanilamide in the colon bacillus infections, especially those infections complicated by pregnancy or some obstructive lesion that can be relieved by surgical means.

To repeat, then, sulfanilamide has had its greatest use in hemolytic streptococcal, meningococcal, gonococcal and colon bacillus infections of the urinary tract.

Sulfapyridine: Soon after sulfanilamide was introduced, it was found that it was of little or no value in the treatment of pneumococcal pneumonia. This led to a search for other derivatives of sulfanilamide that might be useful in the treatment of pneumococcal infections. Approximately two and a half years ago, sulfapyridine was introduced, and we have now had two full years of experience with its use. One can say that this drug is a great advance in the treatment of pneumonia. Our fatality rates during the past few years have shown that in adults, the average fatality rate is in the neighborhood of nine per cent, whereas in infants and children, it is as low as three and a half per cent. These results approximate the best results that were obtained in the past with the use of specific serum.

The patients who respond most satisfactorily, of course, are young individuals without bacteremia, who are treated early and who have only a single lobe involved. Those who have a poorer prognosis, namely, individuals over fifty, with bacteremia or multiple lobes involved, have a higher fatality rate. It is in this group of patients that we continue to use both sulfapyridine and serum.

Our experience shows that patients who have received sulfapyridine and serum have a fatality rate of about 17 per cent. This is somewhat higher than when sulfapyridine alone is used. However, the gross fatality statistics in pneumonia mean very little until you begin to analyze them on a basis of age, bacteremia, and other unfavorable factors. The higher fatality rates in patients who have received sulfapyridine and serum are frequently due to the fact that most of the individuals were over 50 years of age and had bacteremia at the time treatment was begun.

Sulfathiazole: Sulfathiazole has been used during the past year, especially in the treatment of pneumococcal infections, largely because it seemed to be less toxic than sulfapyridine. The most disagreeable side effects of sulfapyridine are nausea and vomiting. We can say, from our own experience, that nausea and vomiting are less frequent following sulfathiazole, that the drug is absorbed more rapidly, and it is excreted by the kidneys quicker, than sulfapyridine. There is less acetylization of the drug. All of these points are favorable, certainly, in preventing side effects which are so common in sulfapyridine, and the results of treatment with sulfathiazole are the same as those following sulfapyridine.

I can perhaps illustrate the course of events that takes place following the treatment of infections by discussing a series of cases which show the clinical course.

Hemolytic Streptococcal Infections

Pneumonia and Empyema. In Figure 1, the course of a hemolytic streptococcal pneumonia with empyema is shown.

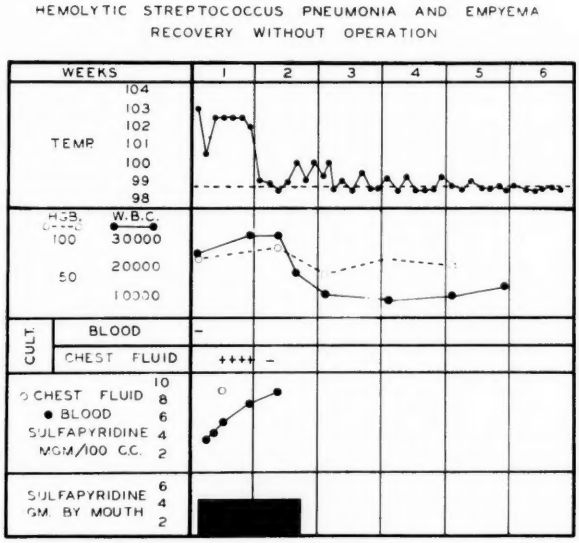


Figure 1.

You will notice that during the first week there was high continuous fever, and within a few days after the onset, there were signs of empyema. Following the administration of sulfapyridine in amounts of 4 grams a day and aspiration of the empyema, the temperature returned to normal and the pleural cavity was sterilized. This was an example of recovery from streptococcal pneumonia and empyema following the use of sulfapyridine without open thoracotomy.

In Figure 2, the course of events is shown in a patient who developed hemolytic streptococcal pneumonia following tonsillitis. There was irregular fever for 16 days, negative blood cultures, and a sterile effusion of fluid into the pleural cavity. Following sulfanilamide the temperature gradually subsided and the patient recovered. In this particular case it was difficult to say whether or not the drug was responsible for the

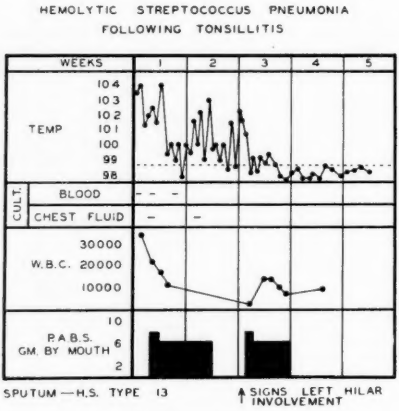


Figure 2.
P.A.B.S. = sulfanilamide

recovery since improvement occurred so slowly. It is true, however, that the pleural fluid remained sterile.

From our experience with hemolytic streptococcal pneumonia it seems clear that, while the disease is not appreciably shortened, the fatality rate is definitely lowered following the use of the sulfonamide drugs. There is no dramatic response in the course of the disease such as is usually observed in pneumococcal pneumonia following sulfapyridine or sulfathiazole.

In the case of hemolytic streptococcal empyema, only about 20 per cent of the patients recover with multiple aspirations of the pleural cavity and chemotherapy. The others require a thoracotomy in addition to chemotherapy before recovery occurs. In some cases, it is possible to sterilize the pleural cavity temporarily but there is often a recurrence of infection when the drug is withdrawn. This is well illustrated in Figure 3.

The pleural fluid in empyema contains substances which neutralize the effect of the sulfonamide so that usually it is necessary to use open drainage of the pleural cavity as well

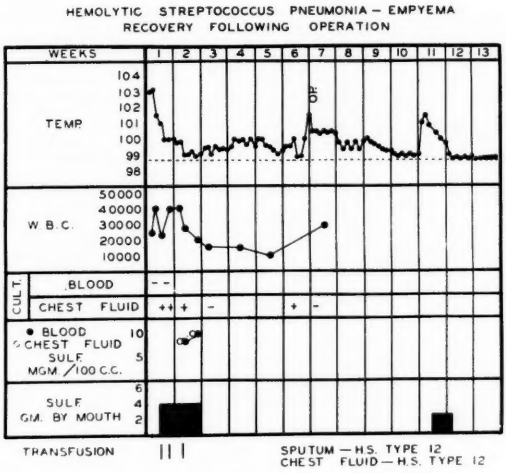


Figure 3.
Sulf. = sulfapyridine

as chemotherapy in order to treat these patients most effectively. We have treated 16 consecutive cases of empyema due to the hemolytic streptococcus with only 2 deaths. This is impressive since at least 25 to 30 per cent of all such patients have died in the past.

Bacteremia: It is now known that the fatality rate in hemolytic streptococcal bacteremia can be reduced to between 30 and 40 per cent following sulfanilamide. It is most striking in patients under 40 years of age who have bacteremia originating from a local focus of infection in which there has been a rupture of the local defense mechanism or in which there is an abscess which can be drained. There are several features in the treatment of hemolytic streptococcal bacteremia which must be stressed. First, the drugs must be given in large amounts (10–12 grams) every day, and they must be given continuously. Since such patients invariably develop an anemia it is necessary to give multiple blood trans-

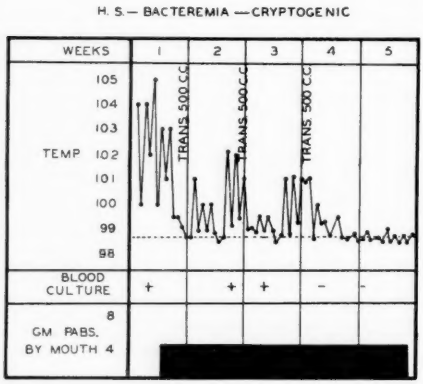


Figure 4.
PABS. = sulfanilamide

fusions to prevent the anemia. Finally, if suppurative foci of infection develop they must be drained at once. In many cases of bacteremia, the blood will continue to show organisms for several weeks in spite of continuous treatment.

Figure 4 illustrates the course of a patient with hemolytic streptococcal bacteremia who had no obvious focus of infection but who had bacteremia for 3 weeks in spite of treatment. Finally he recovered completely without developing any evidence of a localized infection.

In Figure 5, the importance of chemotherapy, blood transfusions and the drainage of foci of infection is shown. A man with cellulitis of the arm developed bacteremia, which was followed by pericarditis, empyema, an abscess of the thigh, and an arthritis of the right elbow joint. Complete recovery followed the use of chemotherapy, blood transfusions, and surgical drainage of the abscesses.

Figure 6 illustrates a somewhat similar situation in which recovery from the infection associated with bacteremia followed chemotherapy and a mastoidectomy.

Figure 7 shows the clinical course in 3 patients with local infection and bacteremia. In 2, the bacteremia followed a tonsillitis. The bacteremia was of short duration and recovery was rapid following the use of the drug. In the third case, death occurred in a patient who had peritonitis and bacteremia.

These cases stress the importance of the location of the

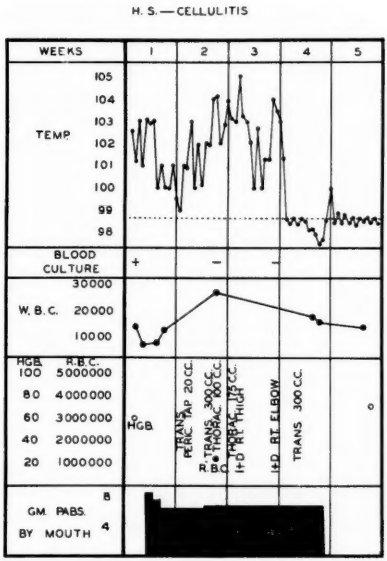


Figure 5.
PABS. = sulfanilamide

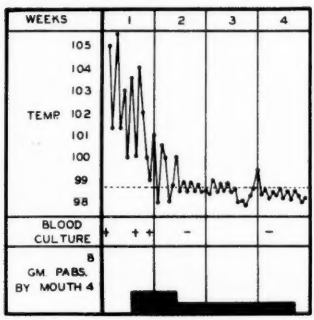


Figure 6.
PABS. = sulfanilamide

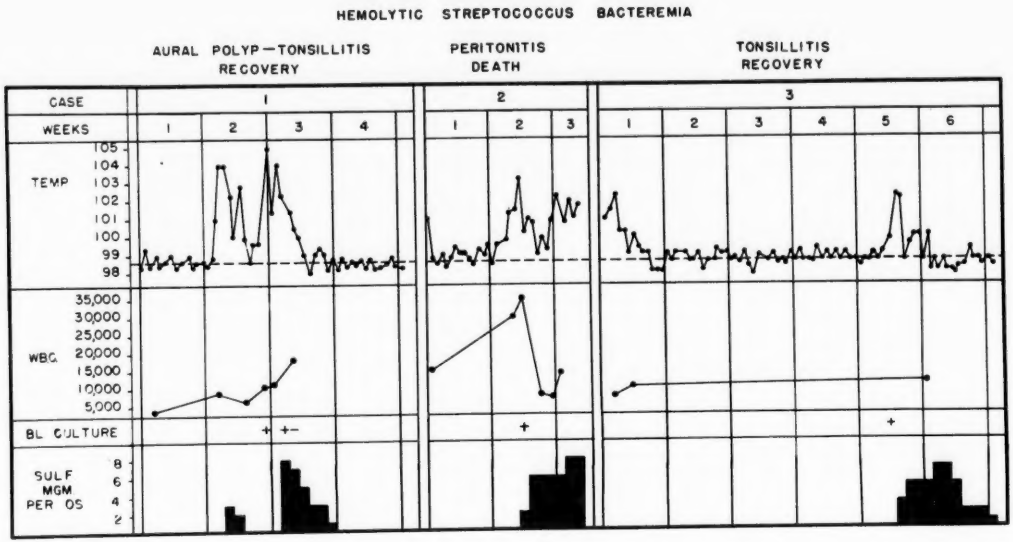


Figure 7.
Sulf. = sulfapyridine

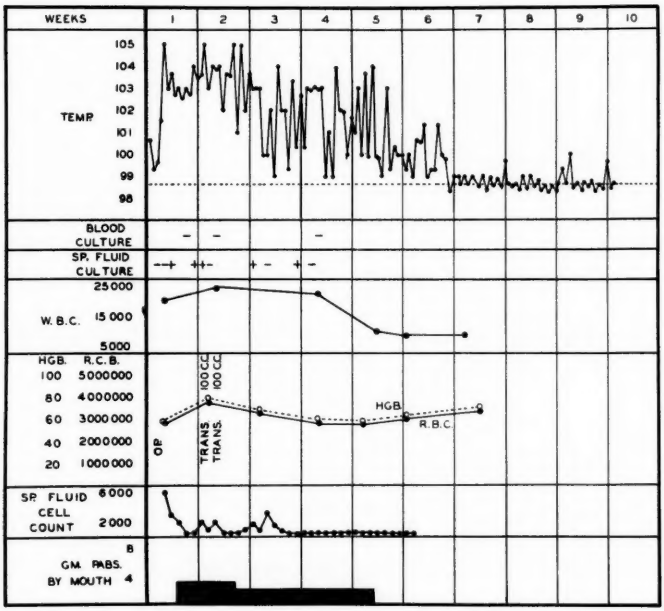


Figure 8.
PABS. = sulfanilamide

original focus of infection in determining the outcome even when sulfanilamide or the other drugs are used.

In the case of hemolytic streptococcal meningitis remarkable results have been obtained with chemotherapy. Before its use, the fatality rate was about 97 per cent. Now, the fatality rate has been reduced to as low as 20 per cent in many series of cases. One should remember, however, that prolonged treatment, with spinal drainage carried out at frequent intervals is an essential part of the treatment. Moreover, if the meningitis arises from the middle ear, mastoid process, or lateral sinus adequate surgical treatment must be carried out at the same time.

Figure 8 illustrates the course of a patient with meningitis following a skull fracture. You will note that this child had irregular fever for 6 weeks and the spinal fluid was infected for 3 weeks. However, complete recovery finally occurred.

One may sum up the treatment of hemolytic streptococcal infection with the sulfonamide drugs by saying:

1. The fatality rate has been reduced in patients with bacteremia, meningitis, puerperal sepsis, and pneumonia with or without empyema.
2. The duration of erysipelas is shortened when the drug is given before the fourth day of the illness.
3. In order to obtain the best results, all other methods of treatment must be used at the same time, such as blood transfusions, surgical drainage of abscesses, and other supportive measures.
4. Finally, the drug must be given continuously and in large amounts.

Pneumococcal Infections

From the evidence now available, both sulfapyridine and sulfathiazole are equally effective in the treatment of pneumococcus pneumonia. Since sulfathiazole causes less nausea and vomiting it is the drug of choice. These drugs reduce both the duration of the disease and the fatality rate. The lowest fatality rates are found in infants and children and in adults under 40 years of age, who have only a single lobe involved and who fail to show bacteremia. While there have been wide variations in the reported fatality rates in pneumonia following the use of these drugs, the average death rate is about 10 per cent for adults and about 3 or 4 per cent for infants and children.

Figure 9 shows the course of a case of Type XII pneumonia with bacteremia in which sulfathiazole was used. It can be seen that the blood was cleared rapidly and that recovery was prompt.

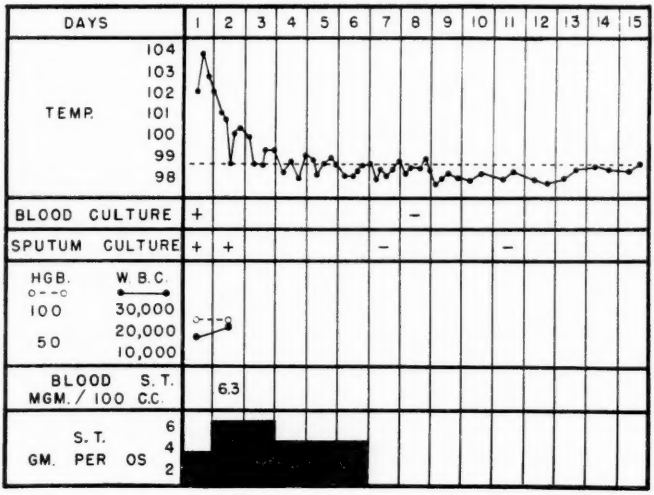


Figure 9.
S.T. = sulfathiazole

Meningococcal Infections

All three of these drugs have been found to be effective in the treatment of meningococcal meningitis or meningococcal sepsis without meningitis. Figures 10 and 11 illustrate the course in two cases of meningococcal infection; one with and the other without meningitis. In both cases the meningococcus was isolated from the circulating blood and in both cases a prompt recovery without complications followed.

The most recent reports indicate that the fatality rate in meningococcal meningitis may be reduced to between 3 and 12 per cent depending on the age of the individual, day of treatment, and the general condition of the patient. It is well to point out in connection with the treatment of meningococcal

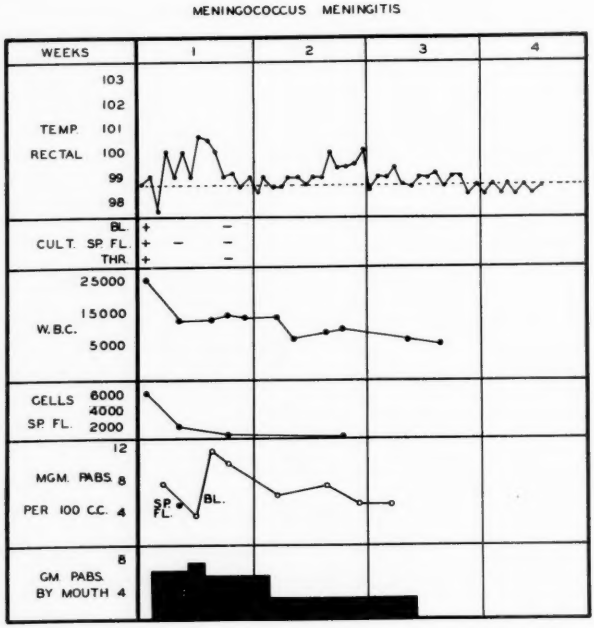


Figure 10.
PABS. = sulfanilamide

meningitis that if sulfathiazole is used, large amounts should be given in order to insure an adequate concentration of the drug in the cerebrospinal fluid.

Gonococcal Infections

The sulfonamide group of drugs is more effective in the treatment of gonococcal infections than any other chemotherapeutic agent. From the information now at hand, it is fair to say that sulfapyridine and sulfathiazole are more efficient than sulfanilamide. In between 30 and 70 per cent of

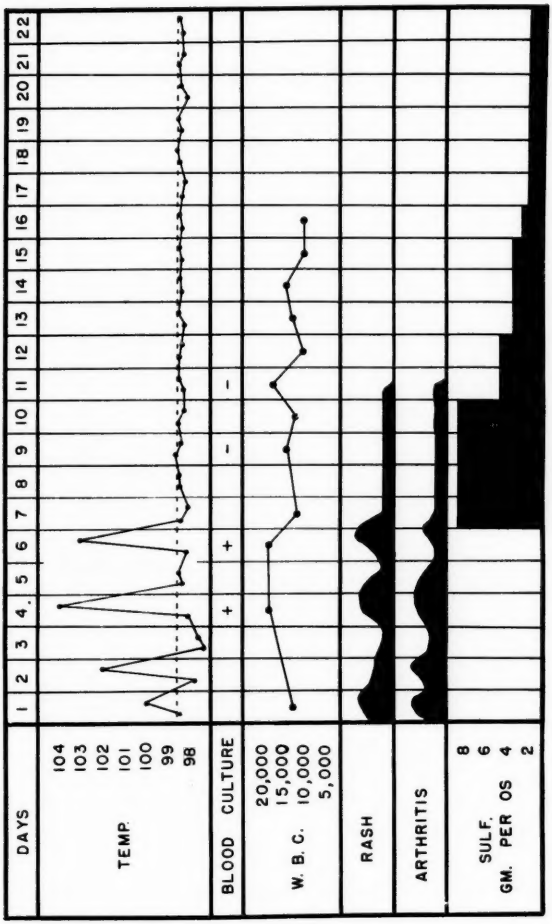


Figure 11.
Sulf. = sulfapyridine

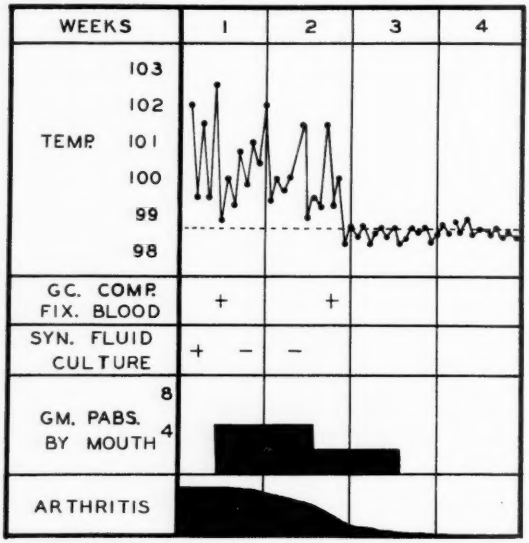


Figure 12.
Gonococcal arthritis — infected synovial fluid.
PABS. = sulfanilamide

cases of gonococcal urethritis the infection can be cured within 7 to 10 days. However, there are several features of these infections which must be stressed; namely, *no patient should be discharged from supervision until the local lesion fails to show micro-organisms after bacteriologic culture.* It is a common observation that patients with gonococcal infections show a marked improvement in the discharge without sterilization of the local focus. These individuals continue to be infectious and unless the proper precautions are taken a sense of false security develops.

When gonococcal arthritis is present, it is known that the synovial fluid may be sterilized within 48 hours in many cases. Indeed the best results would appear to be obtained

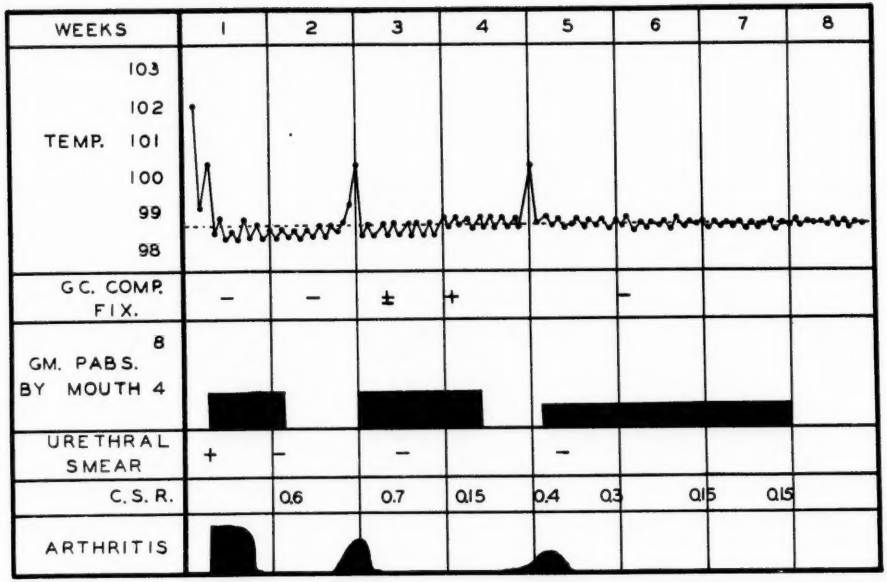


Figure 13.
Gonococcal arthritis — relapses following incomplete treatment
PABS. = sulfanilamide
C.S.R. = corrected sedimentation rate

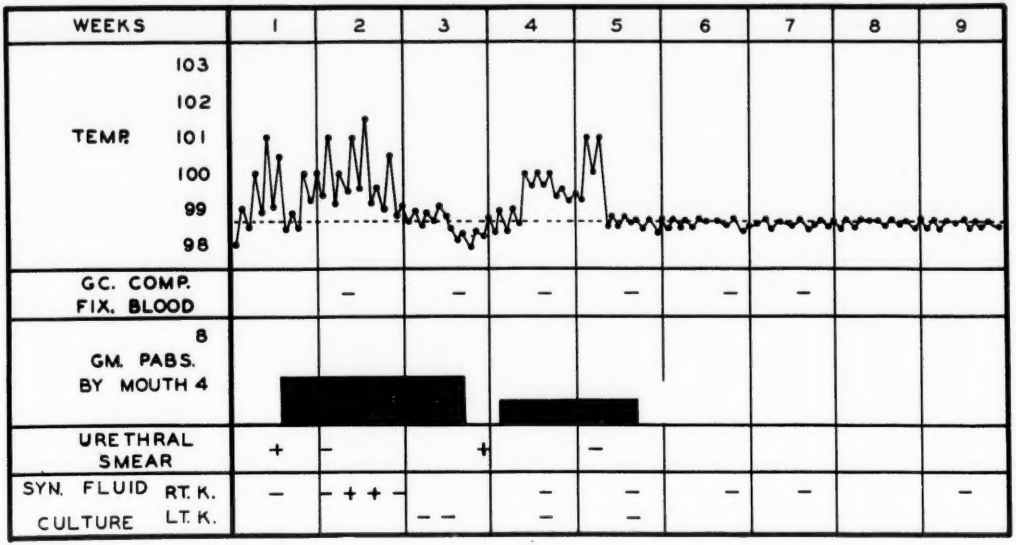


Figure 14.
Gonococcal arthritis — uninfluenced by treatment
PABS. = sulfanilamide

in such patients. In some individuals the infection is very resistant to the drugs and may relapse every time the drug is discontinued. In a third group, there is very little evidence that the drug has any effect whatsoever. Figures 12, 13 and 14 illustrate these three different types of response.

In Figure 12, it is seen that the temperature subsided after a period of two weeks. The synovial fluid was sterilized within 48 hours, the arthritis disappeared within 18 days, and complete recovery followed within 4 weeks. This is an example of an excellent result following the treatment of gonococcal arthritis with an infected synovial fluid.

The relapse of arthritis following the withdrawal of the drug is shown in Figure 13. One can observe that there were two relapses with fever and a recurrence of the arthritis on two occasions when the drug was discontinued.

Finally, in Figure 14, it is seen that recurrent effusion of fluid into the knee joints occurred over a period of 9 weeks, in spite of the fact that large amounts of the drug were given early in the course of the illness.

These three figures, then, illustrate that each case must be followed carefully in order to determine both the course of the disease and the effectiveness of the treatment in gonococcal arthritis.

Urinary Tract Infections

The treatment of urinary tract infections has been greatly changed since the use of the sulfonamide drugs. The main reasons for these changes are that a high concentration of the drug may be obtained in the urine following the exhibition of a relatively small amount of the drug by mouth, and the drugs are effective in either an alkaline or an acid medium so that one need not be concerned with the problem of changing the reaction of the urine.

The most promising results have been obtained with sulfanilamide and sulfathiazole in the treatment of infections due to B. coli, B. proteus, B. dysenteriae, B. influenzae, and

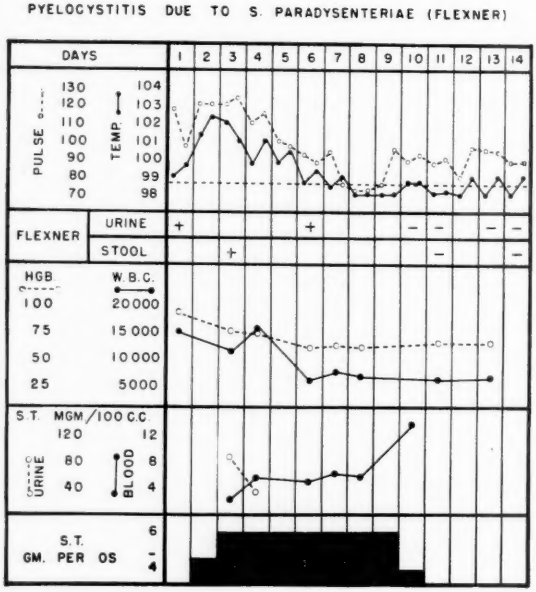


Figure 15.
S.T. = sulfathiazole

Staphylococcus aureus. This is especially true when there is no obstruction to the urinary tract. When an obstruction is present, chemotherapy is useful in preparing the patient for operation to relieve the obstruction.

In Figures 15 and 16, examples of urinary tract infections due to the dysentery bacillus and B. proteus are shown.

In the case of the B. proteus infection, complete recovery occurred only after the incision and drainage of a perinephric abscess which developed during the course of a pyelonephritis.

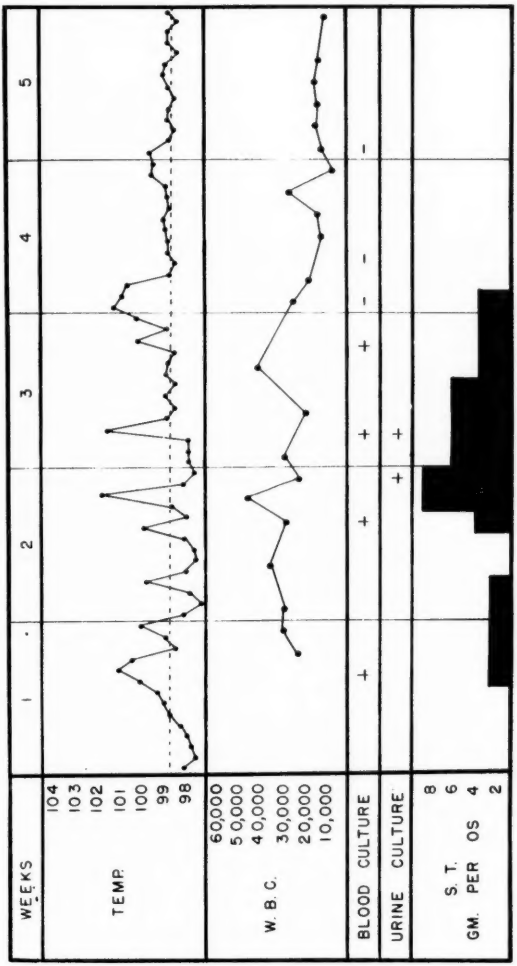


Figure 16.
S.T. = sulfathiazole

Staphylococcus Aureus Infections

These infections are always serious especially when they are associated with bacteremia. Skinner and I (1) found that the fatality rate in bacteremic cases was 80 per cent and that recovery was most likely to occur in patients under 40 years of age who had foci of infection, such as osteomyelitis, which could be drained surgically. Recently Rammelkamp and I (2) have reported 4 recoveries in 7 cases of bacteremia following the use of sulfathiazole. The clinical course in 2 patients who recovered and in one who died is shown in Figures 17, 18 and 19. In one patient (Figure 17) there was bacteremia and a small localized empyema. The blood stream was cleared of bacteria and the empyema fluid was finally sterilized without open thoracotomy. The fever continued for 5 weeks and then recurred following the discontinuance of the sulfathiazole. Finally, an abscess of the right thigh was incised and drained and the patient recovered completely.

Figure 18 illustrates a patient who had bacteremia with a metastatic arthritis of the sternoclavicular joint. The treatment with sulfathiazole and the drainage of the infected joint was followed by recovery. A skin eruption and fever, which were attributed to a reaction to the drug, occurred on the sixteenth day of treatment. Here again, recovery occurred only after the local infection had been drained.

In Figure 19 the clinical course of a patient with bacteremia and multiple foci of osteomyelitis is shown. The blood stream was cleared during the first two weeks, but the local foci of suppuration could not be sterilized by either surgical drainage or chemotherapy.

From our studies of Staphylococcus aureus infection it seemed plain that sulfathiazole is slightly superior to sulfapyridine, and that the best results may be expected in young individuals during the period of invasion of the blood, partic-

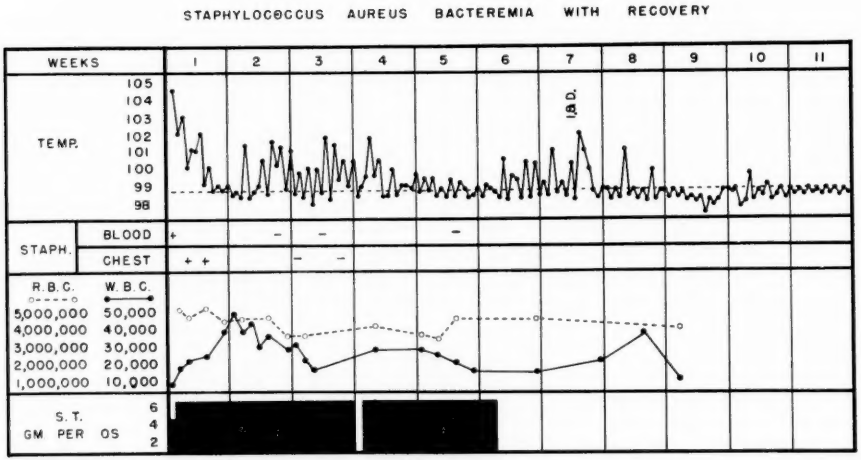


Figure 17.
S.T. = sulfathiazole

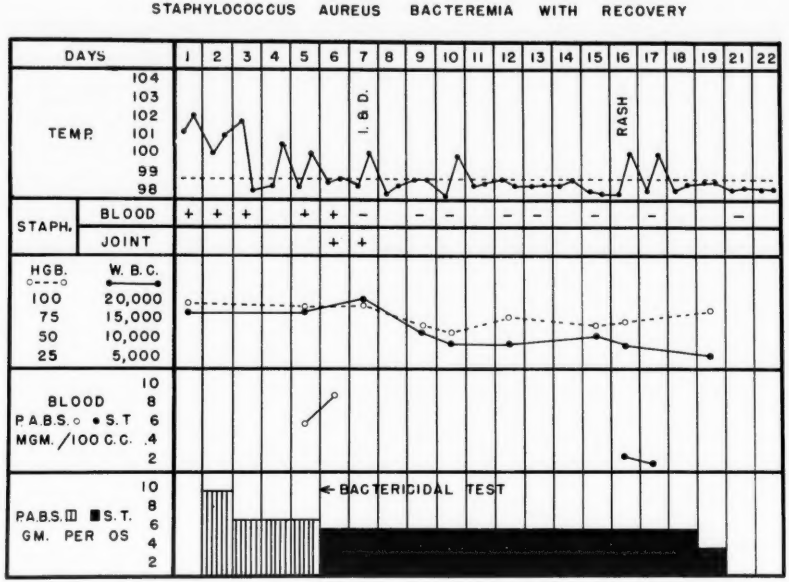


Figure 18.
PABS. = sulfanilamide
S.T. = sulfathiazole

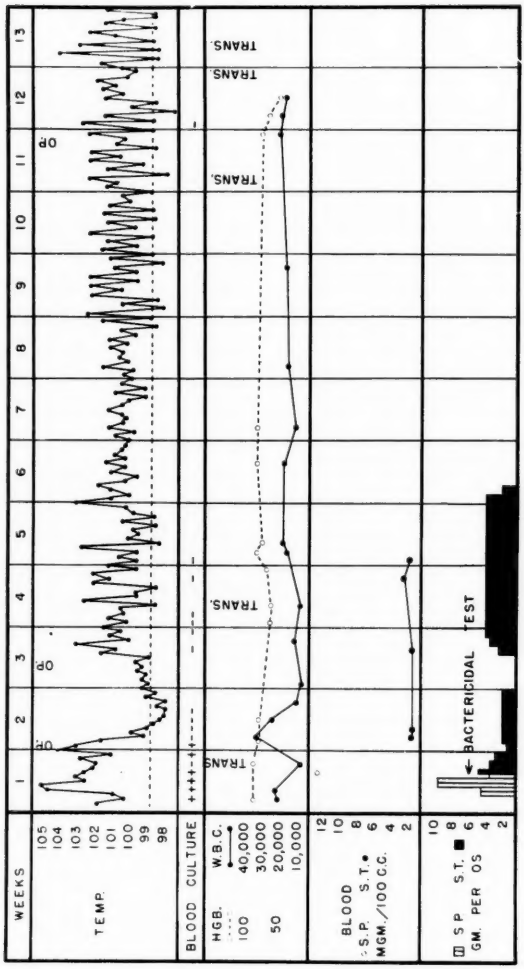


Figure 19.
S.P. = sulfapyridine
S.T. = sulfathiazole

ularly in those patients who have focal infections which can be drained by surgical means.

Summary

I may sum up this discussion by saying that the treatment of infectious diseases has been revolutionized during the past few years and we may look forward to other advances in the near future.

References

(1) Skinner, D., and Keefer, C. S.: The Significance of *Staphylococcus Aureus* Bacteremia. A Study of One Hundred and Twenty-Two Cases and a Review of the Literature Concerned with Experimental Infection in Animals. Arch. Int. Med., in press.

(2) Rammelkamp, C. H., and Keefer, C. S.: Sulfathiazole Therapy of *Staphylococcus Aureus* Bacteremia. New Eng. J. Med., **222**:877, 1940.

President Frost—The discussion on this paper will be continued by Dr. J. Grant Irving, Assistant Medical Director of the Aetna Life Insurance Company. Dr. Irving!

* * * * * *

Dr. Irving—We are indebted to Dr. Keefer for his excellent presentative from the clinician's standpoint of this tremendously important subject.

It has been estimated that 187 tons of sulfanilamide were used in the United States last year. A physician was heard to remark in reference to a well known hospital that all admissions there were now given sulfanilamide routinely. If improvement were not apparent in one week, a physical examination was then performed.

However, even the most conservative appraisal cannot gainsay that progress has been sensational. Those insurance companies who contribute to medical research might well support this fruitful field of study. The advances of the

past few years are perhaps unequalled in the annals of medicine—well named "Sulfa-Miracles"—and yet with new, improved compounds continually appearing, it is evident that the surface has but been scratched.

I shall try to limit my remarks to the insurance significance of chemotherapy under the following headings:

1. Toxic effects in relation to underwriting.
2. Insurance companies' interest in the control of distribution.
3. The influence of chemotherapy on mortality and morbidity trends.

The toxic effects of the various compounds are very similar, and are in general those encountered in poisoning by aniline, to which they are closely related chemically. Those of us who had an opportunity to observe the clinical use of sulfamido compounds at the time of their introduction were impressed and alarmed by the cyanosis, mental depression, nausea, and vomiting. However, time has proven these to be of lesser importance. From the underwriting standpoint we are not particularly interested in such minor effects but we are concerned with serious toxic reactions which may cause death or marked disability. Those complications which assume most practical importance are as follows:

1. Agranulocytosis
2. Anemia of the Acute Hemolytic or Chronic Type
3. Toxic Hepatitis
4. Toxic Psychosis
5. Peripheral neuritis
6. Renal Disturbances
7. Exfoliative Dermatitis

Several questions are pertinent at this point: What is the frequency of serious complications in treated cases? A survey of the literature, including an interesting questionnaire sent out by the Memphis County Medical Society, to which 66 physicians treating 6,522 cases replied, reveals an incidence of 1% to 2%. The type varies, of course, with the drug used; for example: renal complications are much more pronounced

with sulfapyridine and neuritis with sulfathiazole.

Dr. Perrin Long sounds an interesting note here regarding prolonged treatment. He cites an instance where a man with pemphigus has been maintained on sulfanilamide, 2 grams (31 grains) per day, for 23 months. Dr. Long goes on to say that it is his feeling that if a patient does not have toxic reactions within the first two months of drug therapy, it may be continued indefinitely without harmful result.

Now for another important question: How long an interval may occur between the cessation of drug therapy and the appearance of a toxic reaction? In some cases the signs of toxic hepatitis may not appear until as long as 6 or 7 weeks after the last dose of sulfanilamide. This condition may be fatal.

There are cases of agranulocytosis (the mortality is about 50%) reported developing several weeks after cessation of therapy. However, Dr. Long in a personal communication states: "The condition appears between the 14th and 40th day of treatment. As far as I know there have been no true cases of agranulocytosis that developed more than a week after drug administration had ceased". Anemia of the acute hemolytic type usually appears in the first week and if promptly and efficiently treated recovery rapidly occurs. The slowly progressive form of anemia may not appear until the third week of therapy and though recovery is the rule, reponse to treatment is slower.

Splenomegalia may persist for three to six weeks. It is usually associated with hepatitis or anemia.

Renal damage almost invariably makes its appearance during treatment but it is conceivable that it may be missed or progress for an indefinite period before giving rise to signs or symptoms.

In underwriting those who have recently had chemotherapy, then, we should observe the following rules:

1. An adequate waiting period (probably two or three months is quite sufficient).
2. Obtain a Home Office urine specimen.

3. Request a report from the attending physician as to any complications during treatment, especially anemia, jaundice, or renal insufficiency.
4. In special instances or large cases obtain a hemoglobin test and blood smear.
5. Where jaundice or hepatitis has occurred, request a bromsulphthalein or other liver function test.

What of the insurability of those who have had toxic effects from previous chemotherapy? This question assumes practical importance when we recall that once a patient has had a toxic reaction, hypersensitivity appears if a second attempt be made to use these drugs. Remember this would be a small group of about 2% of all treated cases. It would seem that some form of substandardization were in order. The applicant should also be warned to tell his physician in the future of the past sensitivity.

While on the subject of toxicity it is well to emphasize the importance of keeping therapy under control. One has only to recall the 73 deaths in the disaster attendant upon the use of Elixir Sulfanilamide to realize the danger of unrestricted preparation and sale of such potent drugs. Although the toxic agent in that mixture was proven to be the synergistic solvent "Glycol", the following case, which recently came to our attention, will serve to illustrate the hazards of the indiscriminate use of even the pure form of sulfanilamide:

> A white male, age 29, had gonorrhea in 1938 and an unknown quantity of sulfanilamide was prescribed at that time by his physician.
> In January, 1940 he again developed what he thought was gonorrhea, obtained sulfanilamide directly from the drug store, and took 15 grains daily for 23 days. Because of sore throat and skin rash he then consulted a physician and was immediately admitted to hospital. Examination showed a diffuse morbilliform rash over the entire trunk, ulcerative necrotic lesions of the naso-pharynx, and herpetic vesicles on the lips. The blood picture was as follows: R. B. C. 4,700,000 hemoglobin 92%, W. B. C. 600. Differential: polymorphonuclear leukocytes 0, lymphocytes 96%, mo-

nocytes 1%, distintegrated cells 3%.
In spite of transfusions and pentnucleotide injections death occurred 28 hours after admission.
Necropsy showed practically complete loss of all granulocytic cellular elements in the bone marrow, and the final diagnosis was agranulocytic angina.

Legislative acts, as the Congressional Amendment to the Federal Food and Drug Act which resulted from the "Elixir Sulfanilamide Tragedy", and the restrictive laws of New York City and the State of California should receive the wholehearted support of insurance companies. A close watch should be maintained by our Medico-Legal Departments for any attempt to impair the effectiveness of such legislation.

Now let us consider some mortality trends in the years since the introduction of sulfanilamide:

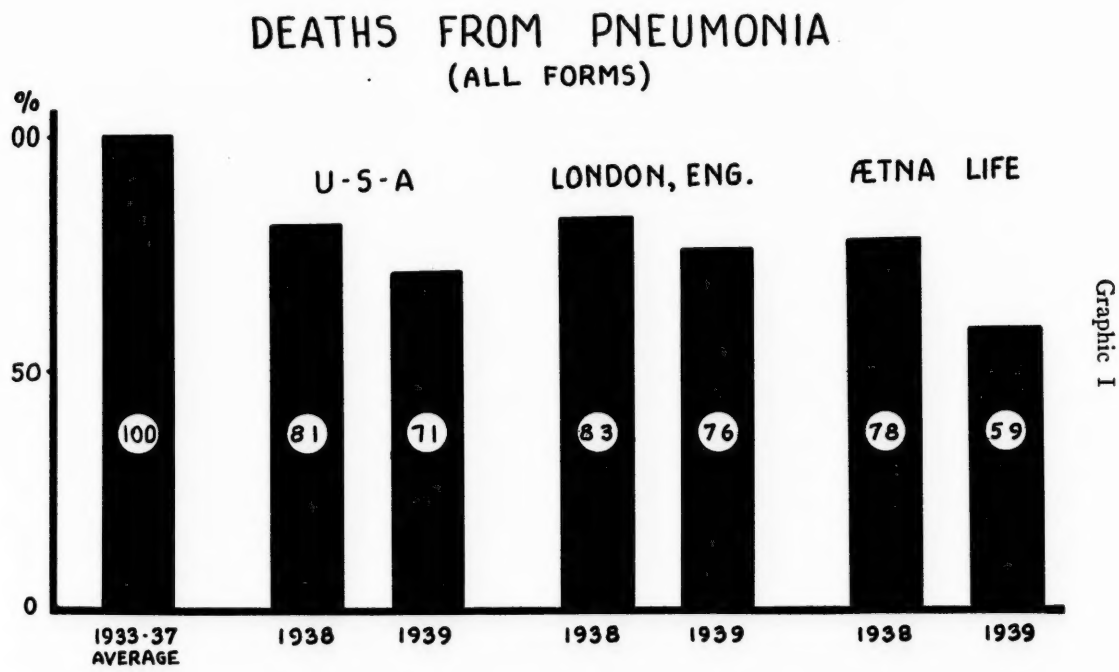
Table 1 represents a review of cases in the literature to date and compares the mortality before and after sulfamido

TABLE NO. 1

APPARENT EFFECT OF CHEMOTHERAPY ON MORTALITY
Review of Literature 1937 to September, 1940

Disease	*Compound*	*No. of Cases*	*Average % Mortality With Chemotherapy*	*Average % Mortality Before Chemotherapy*	*% Reduction With Chemotherapy*
Erysipelas	Sulfanilamide	506	2.5	10	75
Peritonitis—idiopathic of children	Sulfanilamide	8	25.0	54–100	67
Peritonitis—post appendectomy	Sulfanilamide	257	0.4	1.5	74
Pneumonia—Pneumococcal	Sulfapyridine	2709	8.1	21.0*	61
Pneumonia—Pneumococcal	Sulfathiazole	152	7.4	21.0*	65
Puerperal Sepsis	Sulfanilamide	547	5.7	23	80
Meningitis—Meningococcal	Sulfanilamide	1472	10.9	70–90*	85
Meningitis—Meningococcal	Sulfapyridine	174	4.8	70–90*	94
Meningitis—Pneumococcal	Sulfanilamide	34	50	100	50
Meningitis—Pneumococcal	Sulfapyridine	30	38	100	62
Meningitis—Streptococcal	Sulfanilamide	84	20	100	80
Septicemia—Staphylococcal	Sulfanilamide	8	38	60	37
Septicemia—Staphylococcal	Sulfapyridine	10	40	60	33
Septicemia—Strep. Haem.	Sulfanilamide	18	22	95–100	85
Tetanus	Sulfapyridine	22	23	80*	71

*Without specific therapy. Rate with serum varies widely.


DEATHS FROM PNEUMONIA
(ALL FORMS)
%
00
50
0
U-S-A
LONDON, ENG.
ÆTNA LIFE
100
81
71
83
76
78
59
1933-37 AVERAGE
1938
1939
1938
1939
1938
1939

Graphic I

compounds were used. In the last column we see the per cent reduction of mortality which may be credited to chemotherapy. These statistics, of course, are contributed by the leading metropolitan and university centers where treatment facilities are of the best and hence are more dramatic than the countrywide data.

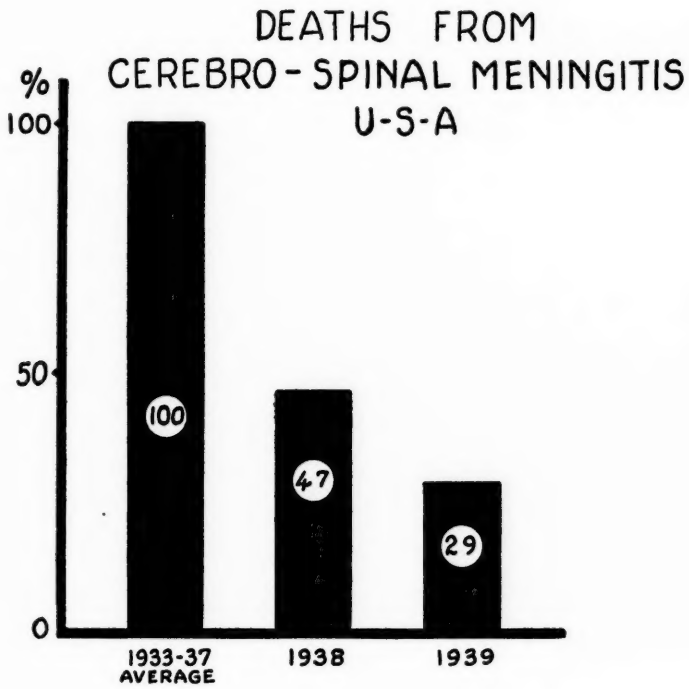
TABLE NO. 2
EFFECT OF CHEMOTHERAPY
MORTALITY

Markedly Reduced	*Improved*	*Improvement Doubtful*	*Unaffected*
Erysipelas	Clostridium welchii	Actinomycosis	Poliomyelitis
Infections, local, of Haemolytic Streptococcal type	Meningitis Pneumococcal	Endocarditis Strep. viridans	Purulent lesions with any bacteria
Meningitis, Meningococcal	Meningitis Streptococcal	Meningitis Influenzal	Rabies
Pneumonia Pneumococcal	Peritonitis Pneumococcal	Plague	Rheumatic Fever
Pneumonia Streptococcal	Peritonitis Streptococcal	Pneumonia B. Friedlander	Rickettsial Diseases
Puerperal Sepsis		Pyocyaneus Infections	Spirochetal Diseases
Septicemia Streptococcal		Septicemia Meningococcal	Trichinosis
		Septicemia Staphylococcal	Tuberculosis
		Tularemia	Virus Diseases
		Typhoid	

However, a consideration of Graph No. 1 showing the crude mortality rate in the United States, London, England, and the Aetna Life policyholders, as well as Graph No. 2 on cerebrospinal meningitis, reveals significant changes which quite possibly reflect the increasing use of these recently discovered methods of treatment.

That the trend in 1940 is still downward is revealed by a comparison of the pneumonia mortality of 90 selected cities, U. S. Public Health reports. From January to June, 1940 this figure showed a 14% reduction from the corresponding period in 1939. Similarly meningitis mortality fell 17% in the same period.

Graphic II


DEATHS FROM
CEREBRO-SPINAL MENINGITIS
U-S-A
%
100
50
0
100
47
29
1933-37
AVERAGE
1938
1939

In Graph No. 3 we segregate the lobar pneumonias from the other forms of pneumonia and note that the improvement is even more marked.

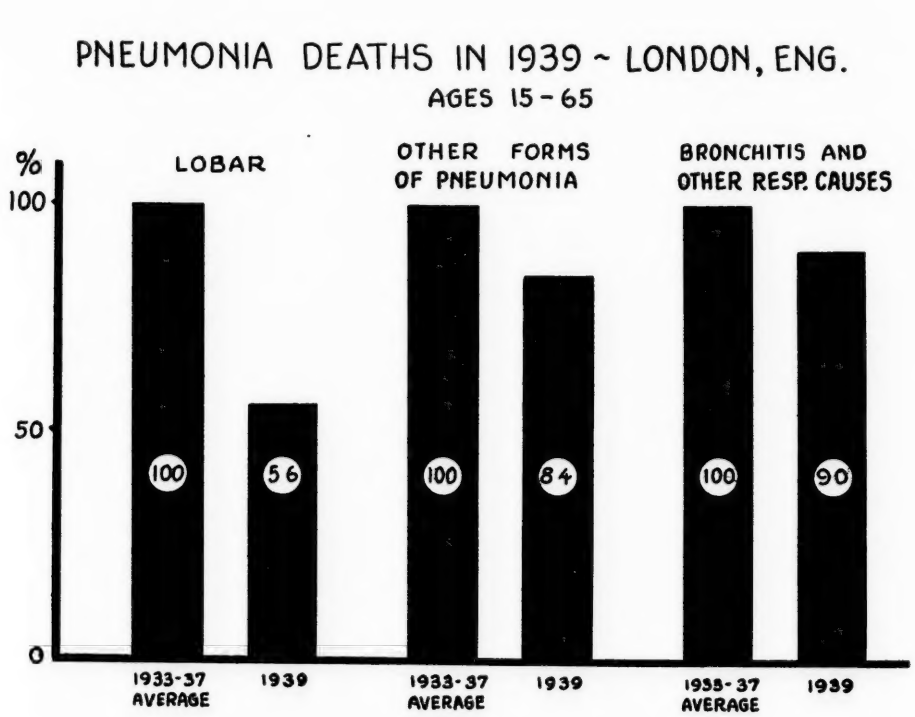
Tables 2 and 3 summarize the effectiveness of sulfamido compounds in the reduction of mortality and morbidity in various diseases.

One might well ask at this time (the day after Draft Registration): What influence will sulfamido compounds have on future military medicine? Here in Table 4 is a summary of all deaths in the American Army in World War I (1917–18). Note that deaths from disease and "Died of Wounds" comprise over 62% of the total mortality.

TABLE NO. 3
MORBIDITY

Markedly Reduced	*Improved*	*Improvement Doubtful*	*Unaffected*
Arthritis Gonorrheal	Chancroid	Colitis, Ulcerative	Arthritis Rheumatoid
Lymphogranuloma Venereum	Dermatitis Herpetiformis	Scarlet Fever	Infectious Mononucleosis
Ophthalmia Gonorrheal	Pemphigus		Fungous Infections
Trachoma	Undulant Fever		Malaria
Urethritis Gonorrheal			Purulent lesions Any bacteria
Urinary Tract Infections (non-specific)			Spirochetal diseases
			Trichinosis
			Virus Diseases

In Table 5 we see the mortality and morbidity of those diseases most amenable to chemotherapy. The British Army reported that 70% of their wound deaths in World War I were due to hemolytic streptococcic infections. Leaving out of consideration the marked reduction in morbidity which could result in gonorrhea, and the controversial subject of acute tonsillitis, let us consider in Table 6 the percent of total deaths due to: Hemolytic streptococcic War wounds, lobar pneumonia, and meningococcal meningitis. Granted that it would be premature and unscientific to make definite prognostications at this time, let us glance at the table and


PNEUMONIA DEATHS IN 1939 ~ LONDON, ENG.
AGES 15 - 65
%
100
50
0
LOBAR
OTHER FORMS OF PNEUMONIA
BRONCHITIS AND OTHER RESP. CAUSES
100
56
100
84
100
90
1933-37 AVERAGE
1939
1933-37 AVERAGE
1939
1933-37 AVERAGE
1939

Graphic III

note that about 1/5 of the total deaths or 37% of those not killed in action in the last War were from diseases amenable to treatment by sulfamido compounds. Now, if we arbi-

TABLE NO. 4
WORLD WAR (1917–18) AMERICAN ARMY

	No.	*Ratio Per 1000 Troops*	*% of Total Deaths*
Deaths from Disease	58,119	14.08	50.94
Deaths from External Causes	5,591	1.35	4.90
Killed in Action	36,694	8.89	32.16
Deaths, Battle Injuries	13,691	3.32	12.0
	114,095	27.64	100.0

trarily apply the improved mortality figures we have previously quoted, it would seem reasonable to hope that 12.2% of total deaths or about 1/4 of those not killed in action may be saved in the next war. For any one particular type of therapy this would be a triumph indeed.

TABLE NO. 5

Infection	*Admissions*	*Ratio Per 1000 Troops*	*Deaths*	*Days Lost*
Meningococcal Meningitis	4,831	1.17	1,836	498,190
Conococcus Infection	251,899	61.02	24	3,903,303
Tonsillitis, acute	176,408	42.73		1,362,609
Pneumonia (broncho & unclassified)	32,572	7.89	9,022	1,233,265
Pneumonia, Lobar	45,774	11.09	10,145	1,845,758

TABLE NO. 6

	% Total Deaths
70% of Deaths from War Wounds	8.4%
Deaths from Lobar Pneumonia and Meningococcal Meningitis	10.5%
	18.9%

SUMMARY

This discussion has indicated the serious toxic effects of sulfamido compounds and how our underwriting practice may be modified by judicious employment of the postponement

period, physician's statement, urine and blood examinations. A plea has been made for aid to further research in this fertile field and the control of the distribution of such potent drugs. Finally, a series of charts and tables has been displayed to indicate the apparent success of the compounds in reducing the mortality and morbidity of certain diseases.

PRESIDENT FROST—Gentlemen, there are just a few moments for questions. If anybody would like to ask Dr. Keefer and Dr. Irving any questions, I am sure they would be glad to answer them.

* * * * * *

DR. HALL—These figures on mortality rates that have been quoted for the general population are very interesting, but they are subject to one fallacy; namely, they do not include the morbidity rate to correspond. I tried to find a morbidity rate for the United States figures, and we don't know what the morbidity is.

The reporting of pneumonia as a disease is not done in a good many states in the union, and they don't figure it in the country as a whole.

I checked up on the state of Connecticut, and found there that the lobar pneumonia morbidity has been practically level, plus ten and minus four, for the past three years; the mortality has dropped, according to the charts shown.

In regard to the interesting things from the underwriting point of view as to the toxic effects, Dr. Keefer mentioned that eleven per cent of the cases given sulfapyridine showed hematuria, and three per cent of the cases given sulfathiazol showed hematuria. I would like to ask Dr. Keefer something about the clinical source of these cases. I think it would be interesting to the Society to know what happens to these cases and whether it is likely that these individuals will have further renal complications that will be of a serious moment in underwriting their cases later.

Incidentally, we medical directors might code such cases, and see whether really they do have serious kidney impairment or not.

PRESIDENT FROST—Are there any further questions or comments?

* * * * * *

DR. PAULI—Life insurance companies are greatly indebted to the clinicians in the various hospitals for the marked improvement in the mortality from pneumonia. We are all familiar with the graph that was published by the Metropolitan in February, 1940, showing mortality from pneumonia and influenza in 1929 as compared to 1939. This mortality curve shows a marked improvement during the winter months, but only a slight improvement in the spring, summer and fall of the year, when compared with the mortality ten years ago.

Does Dr. Keefer find that pneumonia cases treated with sulfapyridine in the summer and fall of the year do not show the same table of results that are obtainable in the winter months?

I should also like to ask Dr. Keefer if there are fewer cases of empyema, following pneumonia, when treated with sulfapyridine than we had with the old treatment five years ago.

PRESIDENT FROST—Are there any other questions? If not, Dr. Keefer, will you kindly close the discussion on your paper?

* * * * * *

DR. KEEFER—Dr. Frost, I want to express my appreciation to Dr. Irving for his excellent discussion of my paper, and I will attempt to answer a few of the questions that have been raised.

In the first place, whether or not sulfapyridine causes permanent renal damage, as far as our studies have gone so far,

one can say that in the vast majority of instances, there is no evidence of permanent renal damage; that is, as determined by the use of such renal function tests. There are instances, however, on record in which, following the use of sulfapyridine, there has been a great depression of the recurrence test and that depression has been present for at least a year after the drug has been given. How often that occurs, I don't think any one can say, because the matter has not been sufficiently well studied.

I do not believe that all patients who show hematuria do so as a result of renal stones. I feel that from observing a number of these patients, there must be some damage to the glomerulus that allows blood cells to filter through into the urine.

So, I think a great deal of study is needed in the future, besides just what the effects are on the kidney, particularly for future damage.

In regard to fatality rates varying in the summer and fall as contrasted to the winter and spring, I have no information concerning the differences in the effect of the sulfapyridine in the various seasons of the year. I think if you studied pneumonia and the results of treatment, you would find that it would be important to break down your statistics on the basis of age and bacteremia. When you do that, you will find that it doesn't seem to make much difference when you get your pneumonia, whether it is the summer, winter, spring or fall. The fatality rate will always be higher if you get pneumonia over fifty years of age, or if you have bacteremia. Those are the two factors, I think, that stand out in any large group of statistics.

Now, that may not account for the reduction in various seasons of the year, but here, again, you must appreciate, perhaps, that variation in type or certainly in complications may be in part responsible for lowered fatality figures at different seasons of the year.

Empyema has been just as common following sulfapyridine as it has been following other agents.

In this respect, I should like to say that we usually encounter empyema in patients who have been treated with serum, who have had bacteremia. If you treat patients early, without bacteremia, you will certainly reduce the incidence of empyema. Now, the reason you see empyema in patients treated early with bacteremia is that you have probably kept them alive long enough to develop a localized infection. Take an individual who has bacteremia, the third day of the disease, and treat him with serum. The blood stream is frequently cleared of organisms, and there is an excess of antibodies and focal infection develops. If, on the other hand, you don't treat patients who have bacteremia on the third or fourth day of the disease, their chances of surviving long enough to develop bacteremia are one out of five, and there is an eighty per cent chance of their dying in the first week of illness. However, if you give them sulfapyridine and then drain the focal infection, patients will recover.

PRESIDENT FROST—Dr. Keefer, we are deeply indebted and grateful to you for your presentation of this subject. It has been very exhaustive, and practically presented, and each one of us is glad to hear the subject.

I want to express my thanks to Dr. Kirkland and Dr. Irving for assisting on the program and giving most excellent discussions.

PRESIDENT FROST—Gentlemen, Dr. Moore has very generously agreed to come and talk to us this morning. He is a very busy man, and I can fully appreciate the courtesy it means on his part to give us this time. Dr. Moore has had such an extensive training in psychiatry and allied subjects that I shall not attempt to detail the various aspects of that training. It is sufficient to say that he is one of our foremost psychiatrists here in Boston. He is a man who is exceedingly versatile. He is a man after my own heart. He is an outdoor man. He likes to tramp and swim and beyond that, he is an author and a poet of note. He very kindly

presented me with a copy of his last publication, "One Thousand Sonnets." Dr. Moore, I assure you I have looked at that book and I have read a good many of them.

Dr. Moore is talking to us today about "Alcohol, A Public Health Problem."

Without further ado, I shall introduce Dr. Moore!

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DR. MERRILL MOORE—It is a great pleasure to be here this morning, because as a practitioner, I appreciate as do many of my colleagues, the important work that you are doing, not only in raising the standards of medical practice but in supplying leadership and stressing the importance of these problems to our national welfare and our social security that every informed person knows today is based upon the services rendered the public through insurance.

I only wish that it were possible that this talk could be more informal, because it is difficult to set down on paper and say what's what about alcoholism, as that branch of medicine is new; in fact, it has only come into the medical fold, so to speak, in the last few years, and because we, as physicians, who study the subject, do not know a great deal about it and are quite confused ourselves about the basic points that concern alcoholism as a medical problem.

There are so many questions and so many statements that I should like to qualify, if it were possible, because I do not want to give you the impression that I, or any one else who is studying the alcoholic individually, or the alcoholics as a group, mean to be dogmatic about it. We are not, and cannot be dogmatic about the alcoholic. And, when it comes to treatment, we are in the dilemma, the situation that doctors found themselves in one hundred years ago, when they were treating medical disorders on a symptomatic basis. That is the way we are treating alcoholism today, because we do not know the basic cause and the diagnosis, at best, is a shaky one.

Those very factors have tended to obscure the whole prob-

lem, and make it much more difficult for physicians to cope with the alcoholic. And, if there is any one here who has not an alcoholic friend, or an alcoholic in his family or among his acquaintances, I should like to tell him that dealing with the problem is very difficult.

It is my opinion, based on rather extensive experience if I do say so, with alcoholics, (because we have 5,000 a year at the City Hospital and many of them or a great number of them have insurance policies) that to treat an alcoholic is as difficult as treating a psychosis. And, alcoholism is as dangerous to the individual who is alcoholic—I mean medically dangerous—as a psychosis; and yet, the alcoholic is not psychotic. Sometimes he is an extremely pleasant, amiable, charming and plausible person.

ALCOHOLISM: A PUBLIC HEALTH PROBLEM

By Merrill Moore, M. D.

To say that you as a group are probably twice as interested in alcohol and alcoholism as most groups may not appear to be a flattering statement. But it occurs to me that you are interested because as physicians you are frequently confronted with the treatment of medical situations complicated by alcoholism and also because you represent the field of life insurance medicine toward which alcohol bears a special but extremely important relation.

Alcoholism is of minor importance as a principal cause of death; but as a contributory factor, favoring the development of organic disease, it ranks high. However, its part as a contributing factor, although recognized, is poorly defined, since no real control studies are possible. Its cost to the individual, the family and the community can not be expressed in simple figures for it involves such intangible and remote costs as the tax rate on your real property, the amount of money available in a tax dollar for schools and roads and the dividends which certain of your well invested securities yield. When one reckons the cost of caring for chronic alcoholics in jails and for acute alcoholics in hospitals, the resulting figure represents only the surface cost. Person with alcoholic psychoses increase the budget of the Massachusetts State Department of Mental Health by 10 per cent; broken homes resulting from alcoholism cause municipal and federal budgets for relief to be greatly increased; the costs incident to accidental deaths which are indirectly due to the intemperate use of alcohol are tremendous. I do not need to point out further to you the vastness of the problem, but I should like to bring to your attention some of the data which we have about the extent

of alcoholism; its social complications and a little of the psychiatric background. In a general sense, the term alcoholism covers the acute and chronic condition as well as the alcoholic psychoses. I shall use the term generally to refer to the entire social, psychological, biochemical and medical problem.

Ethyl alcohol has been used by man since antiquity in the pursuit of pleasure, by well-adjusted people and as a form of escape by others who are either physiologically unable to handle it well or who, psychologically, seem to possess low resistance to its effect and seek euphoria and anaesthesia. The majority of well-adjusted, essentially normal people who drink never come to harm and for one individual who does suffer from its excessive use, there are probably twenty who are unaffected by it and who drink in moderation. I wish to make no brief for the use of alcohol, nor to condemn its use. Its use is a problem which only the individual can solve for himself. I have endeavored to adopt a scientific attitude which aims only at the collection and application of factual data. I hold no brief for the moralistic and regulative attitude nor for the commercial attitude of individualism which denies responsibility after manufacture and sale have been completed. Happily, the groups which hold these two essentially emotional attitudes are compromising their stand somewhat and representatives of the commercial, the scientific and moralistic groups are finding a common meeting place today through the formation of the Research Council for the Problems of Alcohol. This is an organization which seeks through the medium of the Quarterly Journal on Problems of Alcohol now completing its first half year and through other avenues to distribute information about alcohol. It is hoped that educational and therapeutic methods may serve to bring about the end in which the Eighteenth Amendment failed so miserably, i. e. the rational use of alcohol. The scientific attitude is in no wise interested in personal profits but is no less practical for that. The work of the Research Council is growing rapidly and now plans to present a three

day program to the American Association for the Advancement of Science in December. At this meeting the outstanding authorities in America will give a comprehensive summary of the entire field. Representatives of the courts, social service, medicine, chemistry, the distilling industry and the clergy will participate in this symposium.

The fate of alcohol in the body is well understood. About 95 percent of it is oxidized to yield 7 calories per gram and the balance is excreted via the kidneys, lungs and skin. The onset of intoxication varies with the individual but when the blood alcohol concentration reaches 150 milligrams per hundred cubic centimeters, there are unmistakeable signs of intoxication. At a level of 250 milligrams, few persons can walk without staggering and when the blood alcohol concentration reaches 400 milligrams, coma begins. Recovery is rare if the blood level reaches 500 milligrams or 0.5 percent. Of course, it must be remembered that the process of oxidation proceeds at a uniform rate and some is disappearing from the blood as more reaches it from the gastro-intestinal system. So that it is necessary to drink a very large volume in a very short period to reach fatal concentrations in the blood. For the average man, this volume would be more than a pint of pure alcohol in less than an hour. Tolerance and habituation change the time relation of the blood alcohol curve somewhat, although these factors are still somewhat debatable. They seem to bear an intangible relation to personality but this is, at present, a hypothesis, an impression rather than a proven fact. It may be only a chance correlation that an individual with a well organized personality who finds no difficulty in organizing and integrating his behavior is apparently unaffected by a quantity of alcohol which will raise his blood alcohol level to 100 milligrams while the same amount may cause a marked change in a person who is hysterical or neurotic in his make up and badly adjusted socially. Physicians are still considering whether there might be a common physical basis underlying the general personality structure and the tolerance for alcohol. Nevertheless we do see many

patients in psychiatric practice who simply can not drink the smallest amount of alcohol with impunity.

The diagnosis of acute alcoholism is fairly simple. There have been recommended a number of chemical procedures; but, for the most part, these cannot be carried out without the assistance of a trained chemical worker. They require drawing a sample of blood and in medico-legal cases, this cannot always be obtained at a given time. Delay may mean that the blood alcohol concentration has dropped significantly.

In one American city, movies have been made of persons arrested for alcoholism which constitute the record of the accused prisoner's behavior. But there is considerable doubt as to the exact constitutional status of such evidence since no man can be forced to witness against himself.

Dr. Walter Jetter of the Taunton State Hospital has devised a simple apparatus for examining a specimen of expired air by allowing it to pass through a quantity of magnesium perchlorate which absorbs the alcohol somewhat selectively. The tube can be stoppered and analyzed later. In a previous series of control experiments, Dr. Jetter worked out the relation of carbon dioxide and alcohol in the expired air to that in the blood and he now can compute the blood alcohol content accurately from his results. This technique is a modification of earlier methods such at that devised by Harger and Hulpieu in Indiana but has certain practical advantages over theirs. The status of this examination in the courts has not been defined but it has been commended by a number of medico-legal authorities and may someday be acceptable as evidence.

The diagnosis of chronic alcoholism is much more difficult for when a man drinks and is not intoxicated, yet displays the abnormal behavior of chronic alcoholism, the problem is quantitative rather than qualitative. We no longer ask ourselves after a psychiatric examination, "Has this patient been drinking?", but rather, we ask: "To what extent is this man's drinking responsible for his abnormal behavior?" To increase the psychiatrist's dilemma, it is almost universally true that chronic

alcoholism is merely an expression of underlying psychiatric abnormalities and when alcohol is withheld, some other means of antisocial conduct usually is employed as an outlet for the basic drives which constitute the primary disturbance.

The pathology of alcoholism is not definite and many of the lesions traditionally attributed to prolonged use of alcohol have not been produced in experimental animals. Dr. Frank B. Mallory and Dr. Timothy Leary have tried for many years to produce cirrhosis of the liver with alcohol without success. The chronic alcoholic may have gastrointestinal lesions as a result of alcoholic excess but this is not a constant finding. Dr. Robert Fleming has pointed out that the morning vomiting of thick, glairy mucous material so often seen in the chronic alcoholic constitutes the only consistent physical symptom encountered in this condition. It is the only symptom found in almost all cases of chronic alcoholism. It is probable that a lifetime of alcoholic saturation does not favor the maintenance of sound blood vessel walls nor exert an effect like digitalis on the myocardium; but so far, we have been unable to produce significant organic impairment of the cardio-renal system in animals although their reaction to acute alcoholism is in no essential particular different from that of man.

There is, however, one type of disease which occurs in the chronic alcoholic with great frequency, but it is not directly due to the pharmacological effects of alcohol. I refer to the avitaminoses. The life of the usual chronic alcoholic addict or of the periodic drinker (the type who indulges in "sprees") is characterized by a grossly inadequate diet either from the standpoint of nutritional intake or balance. The alcoholic tends to eat very irregularly, if at all, and when he does, his diet consists of high carbohydrate items such as bread, crackers, macaroni, or potatoes. This is in many cases due to the limited money at his disposal but in distributing his expenditures, if he thinks about it at all, he reserves the bulk of his budget for liquor. In the case of chronic drinkers who are not confronted by an economic problem, the foods of choice seem to be those with exceptionally low vitamin and

mineral content. Thus, over a period of time, the vitamin reserves are depleted and are never replenished. It does not require a long period of time for this to occur for we have seen cases at the Boston City Hospital and they have been reported from Bellevue Hospital in which the signs of acute vitamin deficiency have appeared within four weeks after the beginning of dietary imbalance. These conditions included peripheral neuritis, polyneuritis, toxic neuritis, central neuronitis, and Wernicke's disease.

Since it has been possible to quantitate the ascorbic acid content of body fluids, and to relate this to the tissue content, it has been shown that the chronic alcoholic has a subnormal vitamin C level and in many cases a subclinical scurvy has been diagnosed by this examination. In a study made by Drs. Alexander, Pijoan, Schube and myself at the Boston State Hospital several years ago, it was found that patients with alcoholic psychoses had a lower blood plasma content of ascorbic acid than did control patients. The ascorbic acid content in these alcoholic patients was often as low as in scurvy. In view of the fact that some of these patients had been in hospital residence for many years, and considering some other evidence regarding the oxidation of alcohol by the chronic alcoholic, it can be hypothesized that such patients do not have a cellular oxidative mechanism which is functionally as effective (with relation to alcohol) as do non-alcoholic individuals. Of course, other factors than diet may lower the ascorbic acid content, such as infection, but the patients selected for our study were for the most part in good physical health.

The lowering of his vitamin reserves may predispose an individual to have a subdural hemorrhage although the exact relation between the two conditions is not known. In a study of patients who were found at autopsy to have subdural hemorrhages (many previously undiagnosed) the factor of alcoholism loomed large, although some of them had been hospitalized for long periods before death. The records of 3,100 autopsies performed consecutively in Massachusetts

State mental hospitals showed that 7.9 per cent of the patients in this series had subdural hemorrhage. Althaus recognized the relation between subdural hemorrhage and alcoholism as early as 1878 when he observed that the former condition occurred in "old, decrepit and *intemperate*" persons. In the series studied in Massachusetts state hospital, there was an incidence of 83 alcoholic cases per 1000 cases of subdural hemorrhage. No case was under 30 years of age. In these patients, numerous personality changes had been observed.

The principal cause of death in alcoholic patients is seldom alcohol. Other factors are almost always present as complications. The uncomplicated cases account at the Boston City Hospital for less than one percent of deaths among alcoholics. In a ten year study of deaths in Massachusetts due to poisons (undertaken with Dr. Leo Alexander and Dr. Timothy Leary), the records of all deaths certified by medical examiners were reviewed in the office of the Secretary of State. Deaths due to alcohol constituted 52 percent. Although this figure appears large, when it is related to all deaths in Massachusetts from all causes during that period, it is proportionately small. However, it should be pointed out that the causes of deaths as recorded on death certificates are the subject of considerable confusion. There are a number of Standard Classifications of Disease and Causes of Death. The most useful in my experience is that presented by the Bureau of the Census in a pamphlet, "Physicians' Handbook on Birth and Death Registration." It is quite probable that as a cause of death, alcohol is more important than we can demonstrate by the use of statistics, for it is a common and questionably humane custom to spare the relatives of a drunkard the ignominy of such a final diagnosis. This practice is unfortunate from the scientist's point of view.

But I need not tell you gentlemen about that situation for you no doubt are more familiar with it than I am. In the study of deaths from poison in Massachusetts from 1928-1937, we found that a total of 88,090 cases had been certified by

medical examiners, and that 8,861 of these were due to poisonous substances, representing 9.83 percent of all deaths. 4,505 of those deaths from poisoning (or 5.11 percent) were due to ethyl alcohol. If the cases of fatal accident or suicide while intoxicated were added to them, the number reached 4,742 or 5.38 percent of all deaths from poisoning. Thus alcohol not only causes more deaths than any other poison but it causes more deaths than all other poisons put together. Yet, when related to the total number of deaths from all causes, this figure is not excessively large. In the Bureau of Census table for 1937, among the ten principal causes of death (the so-called "killers") alcohol and drugs are not included, yet the ten principal causes cover 72 percent of all deaths for that year, for the entire country.

As a general rule, alcoholics are not prone to suicide and when they do attempt it, they are not especially successful. This may be due to the fact that alcohol may release for some individuals the same self destructive urges that others express in acts of physical violence, such as shooting, slashing or taking other poisons by mouth and its results are less permanent. If it be a method of self destruction, we cannot demonstrate that it is a conscious form and assuming that it may be consciously used for suicidal purposes in a few instances, it is about as ineffectual as iodine. Among 1,147 cases of attempted suicide at the Boston City Hospital, 143 were alcoholic at the time of attempt and only seven or 5 percent were ultimately successful. The average age of both men and women at the time of their attempt was between 30 and 40 years, slightly older than the average for all alcoholic patients but well within the age group of most of the hospitalized acute and chronic alcoholic patients.

The histories of these patients show the breaking of personal adjustments and continued symptomatic drinking often in a periodic pattern or addiction, accompanied by episodes of emotional crisis, often preceded by depression and bewilderment. When sober and recovered, these patients were embarrassed and discouraged and were still confronted and dis-

turbed by various psychological and social difficulties. Many of them were not frankly psychotic but some may have had pre-psychotic personalities or may have suffered from constitutional psychopathic inferiority or hysteria. Most of them were of low economic status but that is true of most patients seen at the Boston City Hospital. We do not have similar data regarding successful alcoholic suicides but there is little to suggest that they are, as a group, very different from the non-successful ones.

I should like to speak briefly on the extent of alcoholism as a hospital problem in Boston. We have discussed and compared the data available for this community with investigators elsewhere in the country and find that the situation, with respect to hospitalized alcoholics, is fairly typical. In the first 70 years of the Boston City Hospital about 50,000 alcoholic patients were treated (or 5 percent of all patients admitted). Yet according to the legal provisions under which the hospital was established, no person was eligible for treatment unless he or she suffered from a medical or surgical condition. In his address at the dedication of the Boston City Hospital in 1865, Mayor Amory remarked: ". . . . .But the respectable poor, virtuous, neat and well-conducted should not be subjected to the daily intercourse with the profligate, who by intemperance and vicious indulgence, have degraded themselves to the level of the brute; whose unseemly habits, profane and rude conversation, would soon drive all others away. For their convenience these apartments were never intended, but both City and State have made other provisions. The government should be firm in declining to receive them, in compelling their removal when inadvertently admitted." Apparently this prudent counsel was found difficult of application, for references to the same problem continue to recur in later Annual Reports. That for 1879, for example, remarks, "But . . . . there are other classes of people who are . . . . victims of their own vices with alcoholism or injuries from drunken quarrels or accidents, who infest the Hospital and whose conditions at the time of application

makes it inhumane to raise any question as to their admission. For all such, certainly there can be no doubt that the City should be repaid under the law, for the cost of their care." (There is, however, no record to date that repayment was made.) A distinct change is observable in the policy of the Hospital in 1879, for the Annual Report states: "The policy of its (the Hospital's) management, as the Trustees are aware, has been the definite one of endeavoring to meet in every way practicable the wants of the public, for whom the Hospital exists, by the admission of all persons who have any claim upon it for medical aid and treatment." After the establishment of the Haymarket Square Relief Station in Boston the same policy was followed theoretically, but in practice, it was never too successful because most of the alcoholics treated there suffered also from complications, as we shall see presently.

There was a steady increase in alcoholic admissions at the main hospital from 1 to 14 percent and the average number was 7 percent for the 70 year period. Of course, there was also an increase in all admissions but the increase in alcoholic patients was usually great as the policy of the hospital became progressively relaxed toward them. Probably during prohibition years, the number was highest but after repeal, it did not fall off to any extent. Male patients have outnumbered female patients 7:1 but this may be due to the fact that women are often cared for at home if alcoholic, where they might otherwise be hospitalized if the pride and shame of the family did not intervene. The average age of the males was 36 to 40 years and that of females was 41 to 45 years. 84 percent were discharged relieved but many of these repeated their hospital entry many times. Those who died in the hospital usually entered in coma and succumbed soon after admission.

On admission, many of these patients were found to be suffering from neurological diseases, especially the neuritides and deficiency diseases such as scurvy and pellagra. Delirium tremens was a frequent occurrence. I do not have at hand the data for the incidence of delirium tremens over the entire

70 year period mentioned above but for the twenty years between 1915 and 1935, 38,376 patients were admitted because of alcoholism and 2,375 of these were diagnosed delirium tremens. This figure represents an annual average of about 250 cases or 6.2 percent of all alcoholics. In 1930, there were 3 percent but in 1935, delirium tremens cases constituted 11 percent of all alcoholics. There is a high rate of fatal outcome when delirium tremens occurs. In the twenty year period, 560 patients with delirium tremens died but the percentage of death has dropped from 52 percent in 1915 to only 14 percent in 1935. In 1932 only 7 percent of delirium tremens patients in the Boston City Hospital died. The lowering of the death rate in this group seems to be related to better understanding of the condition and to improved methods of medical treatment and nursing care. Men predominate among fatal cases and the average age at admission for all male cases of delirium tremens is 36-40 years. For women the average age is 41 to 45.

An evaluation of the causes of death in cases in delirium tremens is difficult. However, two groups seem to stand out; those in which the cause of death is directly related to alcoholism, and those in which the relation is only indirect or coincidental. Pneumonia is a frequent complication of delirium tremens. Studies by Weichselbaum, Grawitz and others indicate that alcoholism and the associated vitamin deficiencies cause a diminution in resistance to bacterial infections. Cases with dilatation of the heart are most frequently found in the vitamin deficiency states associated with alcoholism. These have been proved to be similar to beriberi heart disease by the studies of Weiss and Wilkins. A large number of alcoholic patients die from direct involvement of the brain due either to the neuronitis, pellagra or to Wernicke's disease, which conditions may involve the vital centers of the brain, including the vagus region. Wernicke's disease may be recognizable grossly, particularly if the lesions are associated with hemorrhages, but these cannot be observed in all cases. In the absence of visible hemorrhage, an experienced pathologist may recognize

the bluish-gray discoloration of the periventricular tissue as suggestive of Wernicke's disease. A neuronitis of the pellagra or the beriberi type can be recognized only microscopically. Advanced neuronitis of the vagus nucleus should be accepted as a cause of death, as pointed out by Alexander.

In studying the situation at the Haymarket Square Relief Station in Boston these points became obvious; alcoholic cases which were in need of treatment beyond that which could be given in one day were usually transferred to the main hospital. Male alcoholic patients at the Haymarket Square Relief Station outnumbered females in the ration of 18:1 between 1927 and 1937. Alcoholics constituted 33.6 percent of all male house patients and 13.5 percent of all female house patients. Actually, alcoholics made up only 3.4 percent of all patients because the large majority of patients treated at this institution were suffering minor injuries and were attended in the Out-Patient Department. The average stay of the alcoholic house patient was 1 day and the maximum stay (in one case) was 26 days. Interviews with these patients revealed occupational maladjustment, domestic friction, loneliness and frustration to be the chief reasons they gave for drinking. In the majority of cases, basic underlying personality defects were present and feelings of inferiority which were poorly concealed by the alcoholic episodes. Although the total number of alcoholic patients represented only 3 percent of all patients, the cost of their care was 6 percent of the total hospital budget. This was estimated by multiplying the per diem cost by the number of days of hospital stay. In a group of alcoholic patients treated at the Haymarket Square Relief Station during the 15 year period from 1923-38, (15,229 men and 825 women) 94.9 percent were men and the average age was 40-45 years at the time of admission. 58 percent of the male patients and 25 percent of the women were unmarried. Most of them were born in Massachusetts but of the foreign born group, persons born in Ireland, Canada and the British Isles predominated in that order. The racial predominances probably reflect nothing more than the percentage of these national-

ity groups in the neighborhood. 32 countries were represented as birthplaces. Two-thirds of the patients lived in Boston, many near the hospital and were for the most part, unskilled workers. 12,712 were brought in by the police

Alcoholism was not always the most prominent symptom but each patient had evidence about his person of recent drinking. On discharge, most of them were relieved and were discharged unconditionally but a large number were returned to the police or courts. The chief complications were surgical, but pneumonia, malnutrition, gastro-intestinal disturbances and neurological disorders were common. Many were suffering from frank gastritis and neuritis, cerebral vascular conditions and convulsions when admitted. The most common surgical complications were fractures and head injuries, the latter numbering 10,494 in men and 380 in women out of a total of 16,054 injuries varying in severity from abrasions to compound fractures. Injuries to the arms and legs were next in number and included lacerations, abrasions, contusions and abrasions, contusions and fractures in that order. There were frequent cases requiring treatment for epistaxis, concussion, immersion, dislocations and ecchymoses. Of the medical complications, 517 were neurological and 240 were gastro-intestinal. 138 were cardiovascular and 101 resulted from poverty, malnutrition or exposure. The other body systems were affected but less frequently. The distribution of medical complications was not very different than might be found among non-alcoholics of the same economic level.

The causes of death among these patients deserves brief comment although they numbered only 75 in 15 years (due to the practice of removing serious cases to the main hospital without delay). Only 3 women died at the Haymarket Square Relief Station. All but 7 cases died within 24 hours of admission. Head injuries caused death in 17 cases, and fractured skulls in 15. Cardiac deaths numbered 15. Other causes were concussion, pneumonia, cerebral edema, delirium tremens, epistaxis, malnutrition and starvation, dislocation of the jaw, bullet wounds, burns, veronal poisoning (suicidal), pulmonary

hemorrhage and ruptured liver. There were a few cases in each of these categories.

There was little correlation between the age of these alcoholic patients and their intelligence. It might be said that they were low normal and that their education was limited. The older men seemed to be chiefly laborers and seamen and the men of middle age were salesmen and longshoremen. In many cases there was a failure to make a satisfactory occupational adjustment and a decline with increasing age into unskilled labor groups. Social and economic factors, foreign birth and language difficulty may have qualified such an apparent generalization.

I have gone into these statistics at some length in order to give you a glimpse of the picture of alcoholism viewed as a public health problem which is fundamentally cotangent with your own, and to show you its proportions. In the minds of many of you, there are questions about the treatment of alcoholism and its effectiveness. For the acutely intoxicated person, sedation, the restoration of a normal acid-base and water balance and supportive measures are indicated. These are well understood by most physicians. For the chronic alcoholic addict, only psychotherapy seems to have much value. When there is marked deterioration of personality and the development of psychotic behavior, the patient should be examined by a psychiatrist if possible. In many circumstances this is impossible and psychiatric handling of the case must be undertaken by the general physician. Before psychotherapy is begun, it is essential that the patient should have become free of alcohol following such symptomatic measures as will calm him, enable him to rest, encourage normal elimination and permit him to regain normal fluid balance. The recent memory of the unpleasant symptoms associated with intoxication may be adequate to restrain the patient from drinking for a few days, but memory soon dims, and unless a new program of activity is outlined the cycle of drinking is begun again. The importance of physical well-being as a fundamental step in promoting mental stability is one of the most basic truths in psychiatric thought, and should be utilized at this point. A

number of separate trends in the treatment of alcoholism are now being followed by psychiatrists, and specialists in internal medicine who are interested in the application of psychology in therapy with particular reference to personality factors and the "mind-body" problem. Patients with neurotic personalities and immature emotional development are the ones who respond most favorably to psychological treatment. Those patients with psychopathic personalities who may be pathological liars should not be included in the group considered suitable for psychotherapy, nor should one include patients suspected of being psychotic in the clinical sense. Psychotherapy has not been very successful in the treatment of schizoid individuals who are also alcoholic, or patients in the manic-depressive group. Mental defectives and cases of constitutional psychopathic inferiority are not promising subjects for psychotherapeutics. An unhappy childhood, especially where one or both of the parents have been alcoholic, may predispose the individual to the behavior pattern of drinking. The social and occupational history, the patient's attitude toward himself and to his environment are the best criteria of selection for treatment. For patients whose history suggests deep-rooted, unconscious difficulties psychoanalytic treatment may be more suitable. These cases may be differentiated from cases eligible for less intensive psychotherapy. The intelligence and cooperation of the patient require careful consideration before any psychotherapy is recommended.

A great need is additional hospital space where alcoholics can be treated, especially those beyond the acute state. The attitude of almost all general hospitals is the same as that of the Boston City Hospital; they are grudgingly accepted and discharged as soon as decently possible. We do have in Boston one institution which cares for the alcoholic patient, the Washingtonian Hospital. This institution was organized on November 5, 1857 and incorporated on March 26, 1859. In 1940, it has been reorganized with a consulting and visiting staff of prominent specialists. It is the sole institution

which cares for alcoholic patients willingly, meanwhile maintaining its charges at a level which the average non-pauper alcoholic can pay. Perhaps the greatest value of the Washingtonian Hospital is that it will continue to rehabilitate patients who are gainfully employed and who are valuable to their employers. Except for this hospital, there is no other non-municipal hospital in Greater Boston, for the care of alcoholics except several psychiatric institutions whose charges are very high.

The general attitude toward a chronic alcoholic must embody the concept that his condition is tantamount to a mental illness and that his personality has been damaged at some time in some way. The pattern of drinking belongs in the realm of symptomatology. One of the chief errors in the past has been to consider the problem of drinking as an entity without consideration of the patient's total personality in the situation. His thoughts, emotions and reactions must form the material of therapy. The drinking as a form of behavior may then be evaluated in its true significance and usually as a symptom. In any science a method is valid only in so far as it is appropriate for use with the material under examination. A single chemical analysis cannot be applied for all substances. Likewise one particular method of treatment may be most applicable to one particular type of alcoholic patient. Much important clinical and psychiatric investigation remains to be carried out along these lines in order to answer many questions that arise in this connection. Preventive measures can be easily formulated, but they are difficult to put into practice. A rational program of mental hygiene in childhood along with the warmth of affection and the satisfactions and security of normal family life are the best safeguards against the development of alcoholism. The facing of and adjustment to reality, the building of character and the maturing of a normal personality are more likely to be accomplished under such conditions.

The prognosis in chronic alcoholism is not very favorable. It is not possible to cure alcoholic addiction easily, but with

understanding and cooperation on the part of physician, patient and friends, it is often possible to lengthen the intervals between drinking episodes until they occur only at widely separated intervals. The need for an escape pattern of living can be lessened and when the obsessional features of drinking are understood it is often possible for the patient to make a moderately effective social readjustment.

Parents, husbands, wives, brothers, and sisters find it hard to believe that incorrigible drinkers are not perverse and deliberate. It is difficult to believe that their unfortunate loved ones are relatively powerless to resist the urge to drink. They must realize that the sufferer has a defective ego structure similar to that of a person who unconsciously seeks escape in drug addiction, in some forms of insanity, or more permanently, in suicide. The personality defect is not necessarily incurable, but at best may require months or even years of patient, loving sympathy and support. The aim of treatment is to repair the damaged personality and not by any means to punish the alcoholic. Only when this is realized by patient and relatives is a cure possible.

PRESIDENT FROST—Alcoholism, of course, has very definite life insurance medical angles.

In casting about for a discusser of Dr. Moore's excellent paper, I could think of no one better than Dr. Walter E. Thornton. He needs no introduction to you. I want to take this opportunity to express to him my very particular gratitude because of his assuming this assignment.

Dr. Moore has been extremely busy and Dr. Thornton was not able to get the manuscript until rather late, but he has been a good sport, and, in common with the rest of you, has upheld the Chairman to the full extent of his part.

Dr. Thornton!

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DR. THORNTON—Dr. Frost, in his most scholarly and eloquent Presidential address yesterday, made mention that the first paper ever presented before this organization was concerned

with one important branch of this same subject of alcoholism. That was 50 years ago.

That today's paper should deal with the same general subject is more than an interesting coincidence, it is an evidence that from the beginning this Association has recognized the importance of the problem and has given it consistent and continued study. In this respect we are in line with almost every group professing curiosity as to the nature of things and concern for the welfare of Mankind. The best minds in many fields have concentrated upon it. Dr. Moore represents the neuropsychiatrists; most of the other branches of clinical medicine and of pathology are also concerned as are the biologists, the sociologists, the lawmakers and the jurists, the religionists and moralists, the economists and the political scientists and even the paleontologists and historians. This list is incomplete but is included here in evidence of the striking and unique universality of alcoholism in the history and experience of Man.

The contribution of the medicine of Life Insurance to the knowledge of alcoholism has been considerable. We have made three main classes of studies:

The first is the experience of those insuring organizations which have made premium or dividend distinctions between applicants claiming to be total abstainers and those admitting the use of alcohol. Admittedly the group indices are unreliable and the groups therefore are highly heterogeneous, nevertheless the considerable and consistent excess mortality among the admitted drinkers is impressive.

The second type of contribution is supplied by the great joint studies. The homogeneity here, unsatisfactory as it is, is probably better than in the first group because the information upon which the classifications have been made comes from more numerous and less prejudiced sources.

> The Specialized Mortality Investigation of 1903 threw light upon the subject only as it investigated certain occupational classes in which alcoholism was common. It showed the marked excess mortality

among dealers in alcohol. Of these, those who alleged themselves to be abstainers made a poor showing, but those not so representing themselves were worse and still worse were those associated with the brewing industry. Strange to say, the distillers and their employees were very fair risks, especially in the first five policy years during which they might be considered as almost standard. This surprising effect may be related to the more conservative policy traditional in this industry of not supplying their workmen with their product.

The M. A. M. I. of 1914 records the mortality among policyholders classed according to various degrees and types of indulgence but accepted upon a standard basis. The mortality is in excess of the normal in practically every group and in most of them it is decidedly so. Something of the nature of the excess is apparent in substantial extra deaths from accident, suicide, infections, hepatic cirrhosis and nephritis.

The M. I. S. of 1929 which includes substandard as well as standard risks tells the same story. It demonstrates a particularly high mortality among rated policyholders and it emphasizes the special risk attached to spree drinking where in addition to the causes of death already mentioned, cerebral hemorrhage and organic heart disease were responsible for four times and three times the usual number of deaths, respectively.

It is interesting that these conditions may be due in great part to the associated nutritional deficiency.

The third is made up of the independent company experiences.

Practical difficulties in the study and routine handling of habits cases have been and still are, the interpretation and evaluation of the data upon which the classifications are made and the choice and definition of the class indices.

The information upon which drinkers are classified for insurance purposes can come from five sources: the applicant himself, his friends, associates and acquaintances who contribute solicited testimonials, the agent, the examiner and finally the inspection report.

We can dispose forthwith of the first two of these. Unless the admissions are incriminating, statements from the applicant and character testimonials from his friends and acquaintances can be disregarded. Dr. Moore has aptly pointed out that many alcoholics are pathological liars.

The agent is an interested party and therefore likely to be prejudiced. However, it is encouraging to note that some of them will describe the habits with commendable candor and accuracy. This is a source of information that is well worth cultivating. It has improved and will continue to improve with the rise in quality of the agency forces.

If we were to make the effort I think we might induce the examiner to be a much more valuable source of this kind of data than is the case at present. In the smaller centers the physician is a repository of information of all sorts. If he will tell us of the old lues or of the attack of manic depression, he will also discuss the habits as to alcohol. His training does not lead him to register the common alcoholic excess in the same professional fashion as the physical or mental episode of more definite medical interest and import. He cannot be expected to mention it unless he is specifically and methodically encouraged to do so.

But the classical source of this information is the inspection report. I have had relatively little experience with reports from inspection departments maintained by individual insurance companies and I do not presume to discuss them. What I have to say refers to the reports of the commerical inspection companies.

At this point I wish to pay my particular respects to these reporting organizations. Through the years they have devoted endless study, expert effort and no little expense to improve the accuracy and interpretability of their reports. By the very

nature of their problem the task has been a difficult one but we are now beneficiaries of their labors as any one of experience will realize if he compares the present day product with that of fifteen or twenty years ago. What I have to say of the inspection report is not in the spirit of criticism, rather it is a discussion of the difficulties associated with the reporting of habits cases.

It may be stated at the outset that in practically every case the applicant drinks more and oftener than is reported. If you would satisfy yourself of this phenomenon try ordering reports upon a dozen of your friends with whose habits as to alcohol you are well acquainted. This characteristic understatement is not as disturbing as appears upon the surface because the discrepancy may be regarded as universal. In any one class, the story reported bears a fairly constant relation to the true facts and is a satisfactory index of them. Also it may be taken as an axiom that the more alcohol consumed and the more vicious the type of drinking, the more accurate the report.

A too familiar feature of inspections is the variability among multiple reports. Generally speaking, like blood pressure determinations, they become less critical as they increase in number. The first is apt to be the most accurate because the logical informants tend to become more reticent and evasive on repeat inquiries. Also the most unfavorable report is probably the nearest to the truth because it is more apt to have originated with the one informant or with the few who know the real facts but who have not been found by the more favorable reporters. We do not believe that the often alleged puritanical attitude of informants or inspectors introduces any appreciable bias even in small communities or rural districts, nor do political, personal or other feuds color the picture in any considerable number of cases. The discrepancies are due to varying degrees of understatement and practically never to overstatement.

Sometimes reports show internal evidence of superior quality, as for instance when they relate of specific drinking

episodes in sufficient detail to allow of classification or when they are of the special type made by salaried inspectors. We can afford to be liberal in underwriting such cases in order that our action will be more in line with our handling of ordinary cases in which the habits are understated.

Habits reports from rural areas and small towns are definitely more accurate than those from the larger cities because where everyone knows his neighbors, aberrant behavior is more difficult to conceal and the information is more easily obtained. Inspecting the city man presents real difficulty especially if he is of the more substantial and independent class. Such an applicant is the more important because as a rule his policy is larger than the company average and his mortality is greater by amount than by number. His life is more varied and more complex than that of his rural brother. He moves in a number of worlds that are more or less isolated from one another. His business associates form one world, his club members another, his golf foursome a third, his commuting companions a fourth and so on. It is not practical for an inspection to cover all his contacts and there are many opportunities for drinking which can pass unnoted. Added to all this is the greater tolerance of the metropolitan informant toward alcoholic indulgence. I have no doubt but that some of the excess mortality of large risks is traceable to alcoholic habits which are not unearthed or are only hinted at despite the greatly improved facilities and techniques of the modern inspection procedure.

Turning now to the second of our chief difficulties – the choice and definition of indices suitable for practical classification purposes. These variables differ widely among companies. This results in corresponding variations in the distribution of cases and in mortality in groups which are nominally identical. Because these effects are pronounced, the lumping together of data from many companies is a procedure of doubtful propriety. What advantage is gained by reason of the vast exposure thus obtained, is lost and probably more than lost by the increase in group heterogeneity. The joint com-

mittee itself has strongly and repeatedly emphasized this defect and has insisted that its findings were of only the broadest significance. It expressed the opinion which, I imagine, is now accepted orthodoxy, that so far as habits cases are concerned, the individual company studies are of greater value than are collective investigations.

The Schedule which we follow is this:

1. The *normal social drinker* is the person who constantly and consistently satisfies every social amenity, yet who never imbibes beyond the first or second drink and who never shows definite evidences of intoxication beyond an expanded sociability or perhap the mildest exhilaration. There is no definite evidence of any marked excess mortality present here, indeed the very extent of the group is unknown because few if any companies mark all these cases for special identification.

2. The *social-habit drinker* is the one who appears to wait in sober patience for the social or festive occasion but when it comes he is in the habit of going beyond the mere social requirement and definitely beyond the stage of sobriety. On these occasions he shows the characteristic and familiar alterations of conduct that are clearly diagnostic even to the layman of a material loss of mental and physical control. Note that it is not necessary that the man become stupefied, completely down and out, to classify here. It is customary to subdivide the group according to the number of excesses per year: those averaging not over six, those from six to twelve and those over one per month, usually the week-end drinker. So far as life insurance medicine is concerned, this general social-habit group is by far the most important class numerically for it supplies upwards of 70% of our habits cases. Furthermore, excess mortality is present in all the subgroups and increases with the frequency of the drinking. These social-habit cases provide a rich recruiting field for the heavier drinkers and for the more vicious types of drinking.

3. The *steady free user* drinks every day or practically every day.

My company follows the opinion of the joint committee that the very modest daily use of alcohol, such as two glasses of beer or wine or a single glass of whiskey or its equivalent does not constitute free use. We, therefore, do not include those who follow the continental practice of taking wine or beer with meals. Indeed to us the group does not include "with meal" drinking of any sort. We can claim physiological support for this attitude, for you will recall that in the presence of food elements, especially fats, alcohol is restrained in its passage into the blood steam and produces neither the physical nor mental effects of equal doses upon an empty stomach. We feel concerned only with the drinking that occurs apart from meals.

We belong to what the joint committee would call the "liberal" school of interpretation. We use the time honored Anstie's limit as a general guide. That this index has merit and is useful is demonstrated by its survival for so many years. Dr. Anstie died in 1874. As originally described, it was the daily use of the equivalent of one and one-half ounces of pure alcohol. The M. A. M. I. quotes it as two ounces per day and the M. I. S. follows suit although without naming it other than as "the standard limit of two ounces of alcohol per day".

The man who is known to indulge daily in an eye-opener is a liberal steady free user. So also is he who drinks habitually during business hours. In these cases the classification can be made forthwith without computation as to daily consumption and without the expense and delay of further information.

The excess mortality in the group is substantial but the incidence is very much less than that of the social-habit cases. This incidence varies between 4% and 8% of the total of alcohol habit cases depending upon the types of business accepted. It will not reach 10% even among companies handling substandard, brokerage and reinsurance.

4. The *spree drinker* constitutes from 3% to 8% of the insurance experience among habits as to alcohol cases. He

may or may not drink between times, but on occasions more or less frequent he becomes unquestionably intoxicated and stays that way for days or weeks. In the characteristic case the general pattern of each spree is much the same. Thus the frequency, duration and pattern of conduct, as for instance whether or not he leaves home, are more or less constant for each case.

The inspection report in these spree cases may be assumed to be substantially correct in the description of critical details. This behavior is so far from the normal and the consequences are so marked and far reaching as to leave a trail so definite that the modern inspection routine will find it. Occasionally these drinkers are secretive and of the hermit type so that there may be a good deal of vagueness and mystery about them, but such patterns are not the rule.

This is definitely psychopathic or prepsychopathic drinking and these spreers together with the steadier and freer users are the cases which the physician and the specialist associate with the term "alcoholism". Together they form by far the greater proportion of clinical experience. In other words, the clinical incidence is the precise reverse of that of life insurance experience.

The mortality here is three, four, five or more times the normal depending upon the depth of the intoxication and the frequency and duration of the sprees.

The history of having "taken a cure" enters into some cases. The term "cure" as here used refers to institutional treatment usually of proprietary type. In my experience among life insurance risks, the pyschotherapeutic treatment which Dr. Moore has described is not sufficiently common in the general population to form a group with recognizable insurance characteristics. The observation of such cases from our standpoint is one of the professional privileges yet in store for us. As matters stand at the moment, I believe the chief and indeed the only underwriting significance of a cure as I have defined it is to identify the pre-cured drinking as of the

larger amounts and greater frequency. The type of alcoholism must still be recognized from other data as also the course since "cure" has been completed.

Reform is not without underwriting significance. Like "cure" it identifies the prior drinking as of a degree or type worthy of note and rateable, at least, at the time.

The information almost invariably alleges some specific reason for the reformation. These reasons include marriage, a first baby, indeed the whole gamut of the emotions, the promptings of morality, ethics, conscience or religion, the reactions to economic, family and social responsibilities, the burning lessons of experience (an automobile accident for example, especially if involving a fatality) or the simple dictates of ordinary intelligence.

Whatever the reason, relapses are common though they may be assumed to decrease as the period of reformation lengthens. Provision must be made for the incidence of relapse within the group. We cannot afford to take at face value the confident assurances of the applicant or the happy prognostications of his friends.

Another cause of reform has a different and important significance. This reform is undertaken because the doctor ordered it. The prohibition of alcohol is usual in most of the degenerative diseases. This cause is not frequently admitted but is of such increasing importance after age 40 that reform at these later ages is like the routine physical examination—it almost always means some sign or symptom that is giving concern and that often is of ominous portent. Reform after forty will always bear careful search for concealed impairment.

Mortality in excess of the normal is the invariable experience in all groups of the schedule except the one I have called the "normal social drinker".

It is important to carry in mind the mechanisms of the excess mortality in insurance groups in order that we may arrive at a correct interpretation of our experience. Let us

assume a habits group of perfect homogeneity as to type, frequency and amount of drinking. What we mean is that our group *was* homogeneous at entry. After entry it becomes progressively more and more heterogeneous because some of the risks improve or reform completely; some become worse and only some of them continue without material variation. The first of these—those who improve or reform will yield a mortality below the other two, but these promptly demand reconsideration and in due time are removed from the group. There remains a mixture of those whose habit is unchanged and those whose drinking has become more serious and whose mortality is so much higher so as to weigh adversely and disproportionately in the figure for the whole group. If "A" be the index for entry in a habits group and "X" be the mortality we find for that group, let us not permit the erroneous pronouncement as a clinical fact that a lifetime of drinking to the degree "A" results in a mortality of "X". This is the type of distorted conclusion that throws discredit on our work.

The causes of the excess deaths are instructive.

That violence should be a cause is to be anticipated in this age of automobiles and other machines. It seems clear that the double indemnity benefit should be withheld in all rateable or perhaps even borderline cases.

The suicides indicate not so much that alcoholism causes or predisposes to psychoses as that some psychotics drink in this fashion.

The infections, especially pneumonia, confirm the clinical impression that alcohol depresses the defense mechanisms.

Hepatic cirrhosis and nephritis contribute definitely. There has been a good deal of work upon this question. As Dr. Moore has said it seems clear that so far as experimental animals are concerned, pure alcohol does not cause these states even when given in continuous and all but lethal doses. It is also true that human examples of long continued and excessive alcoholism frequently do not show either of these changes. Never-

theless, the stark fact remains that our groups do yield this excess mortality and from these causes. Dr. Moore has stressed that alcoholism interferes with normal dietary habits. Also alcohol may facilitate transportation of agents that do cause this damage. These tissues in some people for some reason may be vulnerable to alcohol or to the essential oils or other substances commonly associated with it in alcoholic beverages, but we know of no way to select out those who will succumb from those who will survive and, thus, we have no alternative other than to apply proper ratings to all cases at entry.

A surprising feature that is common to most insurance studies of this impairment, including our own, is that the mortality is greater at the younger ages at entry. It is conceivable that the older drinking applicants are the more rugged survivors of general population groups, the more fragile members of which have already died or become disabled. Something of the sort is described in explanation of the effect of alcoholism in improving the average of the offspring in heredity experiments with animals. Also I am of the opinion that at the older ages the groups are less heterogeneous because the drinking habit is then more firmly fixed; there is less drift either toward reform or toward more serious drinking either by amount or by type. Those who do degenerate alcoholwise at these older ages have less physical and mental reserve and elasticity, hence they find the economic road more difficult and policy terminations by modes other than death are disproportionately high. The Haymarket Relief Station's experience supports this contention. Furthermore the older drinker, having lived more years, has a habits trail that is more conspicuous and more easily found and followed because it is longer. Therefore the reports upon him are more accurate. But the main cause is no doubt the relative leniency with which most of us have selected among the younger drinkers and the rigor of our selection among the older. The conclusion is inevitable that the excess mortality extends even to the younger entry years. We cannot under-

write equitably upon the sympathetic assumption that our young drinker is simply an exuberant youth sowing an occasional wild oat, nor upon the indulgent hope that he will steady down with added years, experience and wisdom.

That acute alcoholic poisoning as a cause of death among drinkers should loom much larger than it does in our literature is well known to all of sufficient experience as post-mortem pathologists, coroners or clinical practitioners. The explanation must lie in the defects in the system of reporting the causes of death. Dr. Moore is eloquent upon this point.

There is an impression more or less widespread among medical directors that anti-selection is unusally severe in habits cases and that it increases with ratings or other provisions for excess mortality in such ratio that it never can be overtaken. With this view I cannot conscientiously agree. It is true that in our own experience there has been some excess mortality in the select period and also the not taken rate is definitely advanced but despite our rather liberal policy in considering this type of business neither of these characteristics have been materially out of line with our experience among substandard risks in general including those impaired by reason of purely physical states. Despite the fifty years of experience and study of alcoholism officially represented here, the handling of habits cases obviously remains a problem but I am satisfied that selection can cope with it when and where we know enough about the subject.

One significance of Dr. Moore's paper is that it contributes so freely to our basic knowledge of the subject. We can now go home and build into the structure we call life insurance medicine, the points concerning alcoholism that the doctor has so definitely established for us.

Before I yield the advantage of the floor, I want personally to express to Dr. Moore my warm appreciation of his admirable contribution. It is a welcome addition toward the conquest of a field that, I am sure you will agree, has proven particularly and peculiarly difficult.

DR. S. J. STREIGHT—This Association is deeply indebted to Dr. Moore for the very clear and complete picture of the problem of alcoholism which he has presented.

None of us will find it difficult to agree with his statement: "The majority of well regulated persons who drink never come to any harm". However, our problem is with that minority who may use alcohol to excess.

Individuals who use alcohol in a rational and moderate way, do not find it necessary to take cures, "swear-off", be hospitalized or submit to medical care. Where such a history is developed, we may be certain that the use has been much more extensive than we are given to understand.

When we are advised that the individual has reformed, this is in effect an admission that the previous habits have been such as to call for reformation. If reform is to be effectual, we must make certain that there is the desire, the will and the character to carry out this resolve. Where this is lacking, we may well beware.

As Dr. Moore stated, we are deeply concerned with the social, moral and financial aspects of this problem and the aggregate effect upon the individual and community.

In general, it is difficult to classify these individuals because of the fact that we cannot obtain accurate or dependable information.

Dr. Moore states that the alcoholic is a pathological liar. I am heartily in accord with this and believe that the individual who does drink to excess will never admit the extent of his indulgence. He may not realize it.

Where drinking is of a social character; where there is no tendency to excess, and no pathological cause, there is very little extra hazard. Where there is a predisposing cause, it is impossible to assess its importance or the possible future effect of this habit upon the individual. This is particularly true of the reformed alcoholic.

We will be well advised to reject such lives as these, or at any rate, defer them until time has removed any doubt as to the question of reform.

PRESIDENT FROST—Gentlemen, I dislike to break a promise, but I am doing it on this occasion, and I think you will appreciate my reason. I am not throwing this paper open to discussion from the floor.

Dr. Moore, I am quite sure you would like to say a few things in conclusion.

DR. MOORE—I am afraid we are a little behind on our program this morning. I should like to say a great deal, but in view of my very great interest in coming here, and the most interesting paper that is next on the program, I should like to confine my remarks to just two points.

I hope you will not think that I am riding a hobby horse, when I tell you that the problem of the alcoholic and of alcoholism, I am confident, is more important in relation to life insurance medicine than you may realize. I assume this because I know definitely its importance, as a public health problem.

In view of the medical illnesses directly related to, caused by or associated with alcoholism, it is now definitely established that alcoholism is a public health problem as large as tuberculosis, cancer or syphilis, and it belongs definitely in the category in the medical world with syphilis, tuberculosis and cancer. It is twenty or more times as common and more important as the cause of death than many simpler medical conditions, about which you hear, and very soon we will have some literature available to demonstrate that. The literature is to be printed in the quarterly journal under the Study of Problems of Alcohol.

In closing, I should like to tell you that I feel sure that this group can do a great deal towards furthering what meagre knowledge we have at the present time.

Lastly, I want to thank you for the privilege of being here and for Dr. Thornton's most thoughtful and interesting discussion.

PRESIDENT FROST—Dr. Moore, we are profoundly grateful to you. The reaction of this body obviates any necessity on my part of commenting on the excellence of your contribution, both as to content and matter. I understand you are planning to stay to luncheon with us, and we will have the pleasure of getting better acquainted with you at that time.

We now come to the last contribution to our session.

In 1909, I entered the Harvard Medical School, and, in the course of the next year or two, I came to know a man who was about two years ahead of me. Later on, as an interne at the Massachusetts General Hospital, I came to know him even better. I began to look up to him, for his ability, which then was well demonstrated; not only for his friendliness and charming personality.

Dr. Means and I have been contemporaries in Boston to a certain extent in different fields. We were together more or less during the World War overseas. I don't need to introduce him to you as an expert in the field of endocrinology, particularly with respect to diseases of the thyroid gland; you are all familiar with his publications and his addresses.

Without further ado, I introduce to you Dr. James H. Means, who is now the Jackson Professor of Clinical Medicine at Harvard Medical School, and the Chief of the Medical Service at the Massachusetts General Hospital. Dr. Means will speak for himself!

ENDOCRINE DISORDERS IN RELATION TO INSURABILITY

By James H. Means, M. D.

Jackson Professor of Clinical Medicine
Harvard Medical School

Dr. Frost and Gentlemen: The features concerning endocrinology and endocrine disease that I suppose are of most interest to medical directors of the insurance companies are, first, what effect such diseases have upon health and longevity; and second, to what extent such diseases can be cured or relieved, and third, how permanent such cure or relief may be.

I suppose you want to know how to classify the risk in a person presenting evidence of having, or having had, an endocrine disorder; or, in the case of your insured risks, in which endocrine diseases develop, you want a correct diagnosis made as early as possible so that effective treatment can be given before permanent damage has occurred.

We always want to make early diagnoses, and correct diagnoses, of those diseases especially for which we have successful treatment. It doesn't matter so much if there is confusion or mistake in the differential diagnosis between two incurable diseases. That is rather an academic point oftentimes.

Now, my approach to these questions, perforce, will be that of the physician, not of the vital statistician. I shall give you no statistics. You know much more about statistics than I do. I shall, rather, give you the result of certain clinical observations, and leave you to decide for yourselves what bearing they have upon your own particular problems.

Let us begin with the thyroid, and let us start with those disorders that are due to under-function of this organ. First of all, there is classic myxedema. The diagnosis of this disease is of immediate interest to you. It is often missed, or,

at least, I may say, for a long time it is missed. It is of immediate interest to you because if the disease is untreated, it will cause death in fifteen years or less, whereas with adequate and uninterrupted treatment, so far as we know, the myxedematous subject can live as long as anybody else.

I should like to cite the case of a patient that I have been fortunate enough to have data upon; she is the patient of my friend, Dr. Alexander M. Burgess of Providence. This woman is living now, and she is over ninety years old. She was told, about 1890, that she had Bright's disease, and could not live over six months. The doctor who told her that is probably long since dead. Well, she did live longer than that, and in 1893 she consulted another doctor, who was a better diagnostician. He made a diagnosis of myxedema. He also read his literature and knew that an Englishman named Murray had discovered how to cure this disease, or rather how to relieve it completely. He applied the treatment; he started her on thyroid, and she has been on it ever since. She has a little angina pectoris, at ninety, but nothing to really bother her.

That shows you, at least, that it is possible to live out more than the ordinary life expectancy, in spite of having myxedema.

It is amazing how often this curable disease is missed. We get patients sent into the hospital every year with the diagnosis of Bright's disease, pernicious anemia, and other things, who really have myxedema and who could readily have been relieved in a fortnight, completely, by thyroid.

However, one may make some reservations about this. It isn't always perfectly simple. We got a little high-hat about the diagnosis of myxedema a few years ago, and when a patient came in and said that his doctor had made a diagnosis of myxedema, and when we found that he presented the full-blown picture of pernicious anemia, we thought it was rather too bad the doctor couldn't distinguish between these two diseases. We stopped the thyroid he had been taking, and we put him on liver, and we cured his pernicious anemia; but, in six weeks, he began to get myxedematous. You see, the

truth was he had both diseases, and he required both liver and thyroid to keep him normal. We have since seen several similar patients.

Lately, we have had another reason to be open-minded. We have run into some difficulties ourselves. The problem is not always as easy as we used to think.

We have become conscious of a special type of myxedema, which seems to be really primarily a pituitary disease. Ordinary myxedema is due to primary atrophy of the thyroid, surgical removal or something of that sort, and is entirely relieved simply by giving thyroid, and nothing else. We became conscious of another type, however, by getting into serious difficulties. Certain patients turned up who appeared myxedematous, had low metabolisms, and indeed in whose cases there didn't seem to be a doubt about the diagnosis; yet they didn't do well on thyroid therapy. Some of them on thyroid, although they ceased to have, in a large measure, the classic myxedematous appearance, got sicker instead of better.

To make a long story short, it turned out that these people were suffering from hypothyroidism and also a hypofunction of other glands. Thyroid made them ill because they went into a state similar to that in Addison's disease with adrenal-cortical insufficiency. Two of them went into a state of collapse, and they were revived by abundant fluid, salt and glucose, and in one instance, by the additional use of some adrenal-cortical hormone.

It turns out that these people really have a type of Simmonds' disease, rather than myxedema. They undoubtedly have a lesion of the anterior lobe of the pituitary, which, in consequence, fails to make its thyrotropic hormone in proper amount, so that a hypofunction of the thyroid results. These patients also have hypofunction of other glands, which is not easily recognized because the hypothyroidism occupies the center of the picture, and overshadows the other endocrine abnormalities.

The important thing in these cases is to recognize their nature, because the treatment, necessarily, is different from

that in ordinary myxedema. These patients need thyroid; but thyroid, also, is dangerous to them, unless they are safeguarded by being given proper amounts of salt, or certain other hormones, to substitute not only for the thyroid insufficiency, but the other glandular insufficiencies that are caused by the primary insufficiency of the pituitary.

We cannot, in the time remaining, go into that problem any more. I merely mention it to indicate that there is a special group of patients with myxedema who aren't so simple to diagnose, and who probably carry a less good prognosis.

In real Simmonds' disease, of the classic variety, in which you have cachexia due to atrophy or destruction of the anterior lobe of the pituitary, often caused by thrombosis of the organ, (in women chiefly as a result of postpartum hemorrhage) the outlook is less good than when you are dealing with a primary hypofunction of the thyroid.

The Simmonds' cases usually live not over a decade, or less than that, I should say.

We don't know how long the people with this pituitary type of myxedema may live, because we have not followed any long enough. I expect their life will be shortened, as contrasted to true myxedema.

I should like to say a word about hypothyroidism in childhood. The prognosis in cretins depends almost entirely upon when treatment is started. Untreated cretins may live to be thirty or forty. The cretin is an individual in whom hypothyroidism exists from birth. He is to be differentiated from the patient with juvenile myxedema. In the latter a previously normal child develops myxedema. The prognosis is quite different. If a diagnosis of congenital hypothyroidism, or cretinism, is made in the first few months of life, and treatment started, I think it is possible to get a normal adult, probably with a normal life expectancy, but that happens rather seldom. The diagnosis, often, is not made until a child is three or four years old, and then no amount of thyroid feeding will altogether make good the retardation that has been sustained during the early years of life, when the development

of the nervous system is very rapid and when a retardation, due to an inadequate supply of thyroid, gives you a retardation that can't be made up later by any amount of treatment; whereas the child developing myxedema at the age of ten, let us say, if the diagnosis isn't too long delayed, can be probably made perfectly well again and become a normal adult.

Dr. Frost asked me certain questions; I asked him to do so; items that I might discuss in this talk. He asked, for one, about the significance of low basal metabolic rate, without the clinical evidence of myxedema. Well, that is an interesting question, and we, it is true, frequently encounter people who run minus 20 rates and have either no symptoms or atypical symptoms. I don't think these represent genuine hypothyroidism. We call them non-myxedematous, low-rate cases. We don't know why they run the low rates any more than we know why some people run slower pulses or lower blood pressures than others. Sometimes, they are to be looked upon as simply normal people, who are a little abnormal as to the level of their metabolism only. When they do have symptoms, our experience has been that they may be relieved by thyroid or they may not. If they are not, we stop giving it to them. If they are, we continue as long as it seems necessary.

I don't believe that the existence of such a state of affairs has any very important bearing on longevity, but I can't prove it with figures.

Dr. Frost also asked whether the long-continued use of thyroid diminished longevity. That depends upon for what it is given. It certainly increases longevity in the patients with myxedema, as I have just indicated. Whether some of the people with the non-myxedematous, low-rate syndrome would have their lives shortened by taking thyroid in small doses over a long period of time, I don't know, but I don't believe they would.

Another question is this. How much thyroid can a person take without danger? Dr. Hertz, my associate, tells me he had in the clinic a young woman who took 135 grains of U.

S. P. thyroid at one time, and all she got out of that was a very slight amount of tachycardia. So it is not a very dangerous drug.

Another patient who hasn't myxedema, but who falls in the category of one of these mild, non-myxedematous, low-rate people, has taken six grains of U. S. P. thyroid a day for three years and she seems to think it helps her a bit, but it doesn't change her metabolism; it is minus 15 on thyroid, and it is also minus 15 when she is off thyroid. That is three times as much thyroid as the myxedematous patient gets. What is happening to the thyroid she takes? She either detoxifies or excretes it, or has a resistance to it, or something that permits her to take that dosage.

Let us now take up over-function of the thyroid. This state we can call hyperthyroidism, toxic goiter, thyrotoxicosis, or what you will. Is there more than one variety, and if so, do they have different effects upon health and longevity? That is a controversial question. We can't settle it in the time available. There may be, etiologically, different types of thyrotoxicosis, I shall have to admit. Certainly, we can say that from the point of view of symptoms, we have classic exophthalmic goiter on the one hand, and toxic nodular goiter on the other. Whether they are different, etiologically, I don't know. There are all sorts of intermediate types. One rather sees the toxic, nodular variety in older people, but you can see it in younger people, too.

I have come to the conclusion that there isn't any particular type of person subject to Grave's disease. We see it in old people, in infants and at all ages between, also in males and females, in white, yellow or black, and so on. It is a disease that apparently can afflict any type of person. It is rather rare in Orientals, when they are in the Orient. We had a Chinese who came to this country and got in wrong with the authorities, and landed in Charles Street jail. While an inhabitant of that institution, he developed a classic case of exophthalmic goiter. Apparently, he could not successfully adjust himself to occidental living.

If any of your insured risks develop toxic goiter, you want them cured if possible. This can be accomplished in most instances.

Iodine may produce a remission in the symptoms of any form of thyrotoxicosis. The patient with thyrotoxicosis, given iodine, makes what we call an iodine response. This seems to consist in a diminution in the intensity of their intoxication, which is maintained to some degree as long as they receive iodine and which is followed by an intensification of symptoms, when they omit iodine.

I can show you over and over again the most remarkable parellelism between the intensity of symptoms and the giving or omitting iodine. Indeed, when we deal with a patient with toxic goiter, first we want to know whether they are iodinized or not. Just as in a cardiac, we want to know whether they are digitalized or not.

However, iodine, in most instances, is not a form of treatment, per se; it is used rather to prepare patients for other forms of treatment.

My colleague, Dr. Saul Hertz, has found that there are certain patients with toxic goiter whose symptoms can be totally controlled by iodine and can be kept controlled by iodine, for months, until finally iodine can be omitted without any return.

In other words, perhaps the iodine cured them, but more likely it held the disease in abeyance until it came to its natural termination in recovery.

The more definitive or really curative procedures are essentially two:

1. Removal of not less than four-fifths of the thyroid surgically, or
2. The use of a sufficient dosage of irradiation to the thyroid, usually in the form of X-ray.

Now, I have asked myself the question: What relation does the type of disease, or the type of treatment, have to subsequent health or length of life? It is our belief that if a person is really cured of toxic goiter, he or she may live to a ripe old

age. Certainly, I have seen many who have done so. Also, I think it is fair to say that when X-ray effects an apparent cure, then its result is as permanent as that of surgery. Indeed, I think it may be more so.

That doesn't mean, however, that X-ray is a better form of treatment. Surgery is more likely to bring about a prompt restoration to health than is X-ray, and X-ray fails in a certain number of cases. You cannot tell when you begin whether X-ray is going to cure them or not. But when it does cure them, they apparently stay cured. We follow patients for twenty-five years whom we treated twenty-five years ago with X-ray, and many of them have stayed well. A few of them have subsequently developed myxedema. Relapses occur after surgery, occasionally. Moreover, I think they occur more often than after X-ray treatment.

However, in favor of surgery is the fact that they get well quicker and most of them stay well. The results of surgery are extraordinarily good; they are better than, theoretically, they should be. After all, this disease is presumably due to some influence acting on the glands from outside, and simply taking out four-fifths of a diseased gland, and leaving one-fifth in, shouldn't really cure it. All patients might be expected to regenerate thyroid tissue and relapse. But the remarkable thing is that most of them do not. In some way, the taking out of a big mass of thyroid breaks a vicious hormonic circle of some sort, and permits the individual to establish a normal hormonic balance again. Sometimes, they relapse, however, and then iodine may be used to correct residual symptoms and it is often sufficient.

Nowadays, for recurrent and relapsing cases, we generally use X-ray before resorting to further surgery. Oftentimes, that will take care of the residual thyrotoxicosis and restore such patients to health.

There should be mentioned in connection with all this, something with reference to the late effects of treatment, and the effect of this disease upon longevity. It is certainly true that

in olden times, before modern methods of treatment were available, patients got over this disease without any treatment. We used to see people with prominent eyes, who had had Graves' disease years before, but who were perfectly well, except for a residual of exophthalmos.

I would say, from your point of view, that the fact that an individual had Graves' disease, even leaving him with slightly poppy eyes, was not an indication for classifying him as a very bad risk; perhaps not a perfectly good one, but certainly a fairly good one.

I want to mention, in passing, a subgroup within this field that has interested us greatly within the last three or four years. I don't know whether it is of direct interest to you or not. It probably has nothing to do with longevity or with general health. It has rather to do with the eyes. A subgroup in which the eyes constitute the major problem is something which has greatly interested us during the last three or four years.

In ordinary Graves' disease, the exophthalmos doesn't injure the eye, and, after treatment of the thyrotoxicosis, the eye manifestations and the thyrotoxicosis are ameliorated together. In other words, they run parallel; as the patient gets over his thyrotoxicosis, he also gets over his exophthalmos without any injury to his eyes. In the special cases in question, however, the eye signs seem to have become divorced from the thyrotoxicosis. They vary independently.

What I mean to say is this. A patient has a mild thyrotoxicosis, and gets operated upon for that; whereupon, although his thyrotoxicosis is gone, his eyes proceed to get worse. They become the major problem and a serious one at that. His eyesight is in danger. Indeed, his eyes, themselves, are in danger. Because of the possibility of infection, even his life may be endangered by what we call malignant exophthalmos.

Now, why, in certain cases of Graves' disease, the eyes behave in this atypical fashion, we don't know. However, we have certain views as to how such cases should be man-

aged, and of the significance of the group. For example, we have come to believe that when one can recognize in any given case that the eyes are going to be of the malignant sort, it may be unwise to operate upon the thyroid, because that operation upon the thyroid may make the eyes worse.

Now, all of this must be said with a good deal of reservation, as being in somewhat undeveloped territory and subject to change without notice from the point of view of opinion as to management, but at the present time, we feel that in these cases, giving thyroid may be more beneficial to the eyes than removing the thyroid gland. There is some reason to believe that the eye signs are the result of an excess of the hormone of the pituitary, and that thyroid hormone tends to inhibit the pituitary. When the exophthalmos is progressive, some of these cases have to have the orbits decompressed by a neurosurgeon.

I must not go on longer with this particular subject. It may not be of great interest to you. But I think that you should be conscious of the special subgroup, in which the eyes are the major problem, and are in danger, and in which the patient is not in danger, because the general management is quite different, and because, after all, you want your risks to get the right kind of treatment. They may be treated incorrectly by one not familiar with the special type I have been discussing.

I ought to talk to you about malignant goiter and what that has to do with life expectancy. Nodular goiter may be a precursor of cancer, but whether all nodules in the thyroid should be removed as soon as discovered, I can't settle. It it a controversial subject.

In surgical clinics, one is told that most nodules should be removed. Certainly most of them get removed. Physicians, however, are more conservative. I can only tell you that in our clinic, prophylactic thyroidectomy on non-toxic goiters hasn't always prevented cancer. I can also tell you that if all nodules were taken out, it would mean that most of the population would have to be thyroidectomized.

Now, I think I have about ten minutes left and all I can do is to give you a catalogue of certain other endocrine matters that may be interesting to you.

Dr. Albright, at the Massachusetts General Hospital, as you probably know, has been very much interested in the parathyroids. They should interest you because of the serious diseases connected with them. Some can be successfully relieved, if not cured. You want to know about that in connection with any insured risks who may develop this disease.

Hyperparathyroidism occurs in two forms; hyperplasia of the entire system or a hyperfunctioning tumor. The hormone of the parathyroid pulls out calcium from the skeleton, so that it is depleted of calcium and bone cysts develop, with spontaneous fractures, and so forth. That is one result. Another result is that all this extra calcium going out through the kidney may damage the kidney. It may give rise to kidney stones or diffuse calcinosis of the kidney, which impairs function. Thus these people suffer renal injury, which, if the thing is allowed to persist too long, may be irreversible. Therefore, the early recognition of this disease and operative treatment, the search for the tumor and its removal, if it can be removed, before the kidneys sustain too much injury, is what should be done. If it is done, the patient may get a good result.

I should like to lay before you the thought that any endocrine gland may hyperfunction because of the development of neoplasms. Theoretically, any of these conditions, whether of the parathyroid, the adrenal cortex, or of the islets of the pancreas, may be cured if the tumor can be removed. I might also say that hyperparathyroidism would cause death in a few years, if not successfully treated, but if it is successfully treated, I think you would have a good life expectancy.

There is another disease of the parathyroids, that in which they don't work enough. Individuals suffering from such a defect have attacks of what is called tetany. They have low

calcium in the blood and giving calcium relieves them. There is also a substance dihydrotachysterol, chemically half-way between the parathyroid hormone itself and Vitamin D, which is very useful in their treatment. Patients treated with this agent may have a fair life expectancy.

I am not going to say anything about Addison's disease. You know about that. But there, again, is a disease that was formerly fatal, and which has now, if not a completely successful treatment, at least a treatment which offers a good deal; namely, first of all, giving large amounts of salt, which controls many of the symptoms; and the use of a portion, at least, of the missing cortical hormone, or hormones, which can be given in the form of desoxycorticosterone intramuscularly or in the form of subcutaneous pellets.

Thus if you had had an insured risk a few years ago who developed Addison's disease, the company would have had to pay out, I expect. However, nowadays, the life expectancy certainly has been lengthened. I can't tell you how much it has been lengthened, but certainly sufficiently so that the risk will be able to pay several more premiums.

This whole subject is so interesting that one hates to rush it, but it is necessary. Cushing's disease is another type of adrenal-cortical dysfunction, due either to stimulation by the pituitary or primary hyperfunction by the cortex. People with this disease develop premature senility; they get hypertensive; they have an insulin-resistant diabetes; their skin atrophies; they have a tremendous muscular weakness, their bones become osteoporotic. They lose nitrogen. Except when a tumor could be found and removed, there was little to be done in these cases until recently. Albright has lately shown that the male sex hormone, testosterone, even in females, may confer marked benefit. This hormone seems to stop the nitrogen loss and improves the general condition. It may prolong life, but I don't know how much.

I was asked particularly about dwarfs and giants. Perhaps it will be best to speak of these briefly, and then stop. Indeed, I don't know exactly when an individual can be called a giant.

You know that better than I do. But I would say that a real giant, whose disease is due to over-supply of the growth hormone of his pituitary during the growth period, and at the same time usually with a diminution in the supply of other hormones, has a bad prognosis. They don't live to be much over thirty, usually. They pick up some infection like tuberculosis, and die.

Now, the individual whose epiphyses have united before the pituitary becomes over-active, doesn't become a giant; he can't grow in height because the epiphyses have been closed; he can grow in other ways, so that he takes on the familiar appearance of acromegaly.

What significance has that from the point of view of insurability? I should think that an individual in the active stage of acromegaly would probably be a poor risk. These people develop intercurrent disease, like tuberculosis, and die of it. I have known that to be so. On the other hand, we do know that sometimes they go into a quiescent phase which may last for many years, and I think that occasionally, if the disease it not due to a tumor, these people may be capable of living to a ripe old age.

I spoke of Simmonds' disease. These people are very emaciated and weak, impotent and so on. They don't live more than a few years without treatment, certainly.

However, a successful treatment for them hasn't really been found as yet. They need the whole anterior lobe secretion. But, some progress is being made by supplying them with the hormones of the various glands under-functioning as a result of the primary pituitary under-functioning. My own belief is that before long, we shall have a successful treatment for this important group of cases. They are mostly women, in whom the disease began following a postpartum hemorrhage, and I should like to throw out the thought that postpartum hemorrhages are important to you, as being the proper etiology of a wasting disease a few years later, which may be of grave significance to you from the point of view of life expectancy. One last thing, this strange disease is imitated

closely by what we call anorexia nervosa. That is to say, the psychotic individual who won't eat, and gets into an extreme cachexia; the pituitary is starved, and a state of true hypopituitarism develops, not due to disease in the gland, but due to a primary psychogenic factor. The treatment of these people is diametrically opposite to that of those who have an organic lesion of the pituitary; it lies in the field of psychotherapy. I think that Dr. Moore will agree with me that they are challenging problems for the psychiatrist, and difficult to deal with. They are resistant to treatment. Some of them are successfully treated, and when so, there is no reason why they shouldn't become perfectly normal people.

It is time for me to stop, I am afraid. I was asked about obesity. As a parting shot, I will suggest that obesity is usually not an endocrine disease, but simply one due to overeating. You probably know more about its effect on longevity than I do.

I thank you very much.

PRESIDENT FROST—I have asked Dr. Daniel Shewbrooks to discuss this subject of "Endocrine Disorders in Relation to Insurability". Dr. Shewbrooks!

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DR. SHEWBROOKS—Mr. President, members of the Association, and guests: Sir Thomas Browne once said–

> "Some have keen wits to know the truth,
> Some have strong hearts to tell the truth;
> But how few know to tell it so
> That all men see it is the truth."

Doctor Means not only knows the truth about endocrinology, but has made us see it through his able presentation, based on his rich clinical experience. In fact, he has shed so much light on the practical side of this interesting and difficult phase of insurance medicine that I, for one, shall look forward to studying the transcribed notes, to profit the more from his wisdom.

Any attempt on my part to discuss a paper so ably presented must, of necessity, be extemporaneous, because, as our President said in his off-the-record remarks, it was not until our guest speaker completed the presentation of his paper, a few minutes ago, that I had any idea of what he was to say. To comment, therefore, on even a portion of the ground covered, is, at best, difficult.

No one excepting an active worker in the field of endocrinology can attempt to follow the enormous amount of literature (physiological, biochemical, and clinical) presented to-day. Even the specialist must restrict himself to a single branch. One is often confused because of the diversity of opinions and conclusions drawn by experienced workers from the same set of facts. A strong position taken to-day is only too frequently reversed tomorrow. We do truly, like Omar of old, come out the same door by which we entered,—little or none the wiser.

To-day, the desk of the general practitioner is flooded with streamlined advertising, outpourings from ambitious pharmaceutical houses, all hoping "to hit the jack pot." Can we wonder if the practitioner is hopelessly bogged by conflicting, often uncritical, clinical reports, which eventually force him to rely on a hit-or-miss trial of therapy with "endocrine" remedies of which he ignores the source and often the dosage! He is forced to and likes to practice endocrinology. We see daily in our work cases which have been treated for this or that so-called "endocrine dysfunction." From a life insurance standpoint, it becomes a very practical and important question, because of the present degree of knowledge of endocrinology among physicians at large, as to how this class of risks shall be evaluated.

It is evident that we must first determine on what scientific basis, if any, the diagnosis of an endocrine disorder was made and what treatment was rendered. This at once throws into sharp relief the qualifications of the physician making the insurance examination, the experience and training of the attending physician, the reliability of laboratory technique,

and, finally, the training and judgment of the reviewing officers interpreting the findings. To judge of the qualifications of the examiner and the attending physician, the various Company records usually suffice. The reviewing officers, however, should have not only a thorough knowledge of life insurance medicine, but also a thorough training in clinical medicine and an intimate familiarity with recent developments in the field of endocrinology. Obviously, cases of this kind should be referred to the particular medical officers who are specialists in the field and alone are able to direct properly development of the history through the examiner and the attending physicians (a procedure frequently more important than the examination itself) and, finally, to evaluate the facts in terms of life risk.

With few exceptions, life insurance statistics afford little practical aid in evaluating the effect of endocrine diseases upon health and longevity. This, the more so because endocrinology is still in its infancy, our knowledge of it is in a marked state of flux, and the risks available for statistical analysis are negligible as to number of lives, volume, classes, and years of experience.

In contrast to much of the foregoing, the thyroid affords an exception to our statistical and medical armamentarium. Physiologists, biochemists, surgeons, and clinicians in this special branch of endocrinology have been and are giving us significant and definite observations and facts, which are of inestimable value in their bearing upon our particular problems of selection of this class of business.

It might not be amiss to recall a few of the fundamentals. The thyroid contains a hormone, thyroxine, the action of which is to stimulate and regulate metabolism. This hormone is formed constantly in the gland, where it exists in combination with protein and through the blood is distributed to other tissues. This action is in some way dependent upon the presence of iodine in the molecule.

Risks residing in regions where there is less than one part of iodine to one billion of water are apt to have endemic

goiter. Their thyroids attempt to supply the full amount of hormone by means of cell hyperplasia, producing a diffuse, soft, symmetrical enlargement of the entire gland. In the later stages of the disease, the nodular type predominates.

The ill effects of iodine-lack are intensified with each passing generation. Yet, adequate prophylactic use of iodine, in the form of iodized table salt and sufficient iodine-containing food, is capable not only of eradicating this form of cervical deformity, but, what is of greater importance, of controlling those forms of physical and mental degeneracy which are dependent upon thyroid insufficiency,—cretinism, mutism, and idiocy.

Life insurance is dependent not only upon medical judgment as to what degree of enlargement of the thyroid shall be termed "goiter," but, what is vastly more important, upon whether that judgment is able to state accurately whether or not there is present an alteration in its function,—either decrease or increase in the activity of the gland.

The condition of myxedema results when a marked degree of thyroid insufficiency occurs either from operative removal of the gland or from degenerative changes. We have in this condition the simplest or most clean-cut endocrinopathy known,—easily explainable on the basis of the absence or scarcity of a single hormone. The difficulty here is that the thyroid cannot make the hormone, not that the ingredients for hormone manufacture are scarce or absent, as in endemic goiter. Since the thyroid hormone plays a role in growth and maturation, the effect of a lack of it during the growth period will be more devastating than when full maturity and growth have been attained. For this reason, there is greater danger of myxedema in children operated for exophthalmic goiter, although the operative mortality in children is no higher than in adults. A child needs his thyroid very much more than an adult does. Thyroid extract serves very well as a substitute for thyroid provided one has attained full growth and mental development. Doctor Means paints a brighter picture for these young people so unfortunate as to develop myxedema

from thyroidectomy, as he believes that, when the condition is recognized early and adequate treatment commenced, they can eventually be made entirely well again and develop into normal adults. Such individuals, of course, do present an increased risk from a life insurance standpoint.

How should risks which have low basal metabolic rates, but no clinical evidences of myxedema, be classified? First, hypometabolism is not synonymous with hypothyroidism, which requires the combination of symptoms and signs of myxedema and a lowered metabolic rate. The Lahey Clinic claims that it has only once seen a patient with a basal metabolic rate of lower than minus twenty-four who did not have clinical evidence of myxedema. On the other hand, the Clinic has seen a large number of patients with basal rates minus ten, fifteen, eighteen, nineteen, twenty, etc., who have been on thyroid extract feeding for long periods of time and who actually are not benefitted at all by thyroid extract.

Doctor L. M. Hurxthal, of the Lahey Clinic, working with blood cholesterol, has demonstrated a valuable method of segregating those patients with low basal rates who have no myxedema from those patients with low basal rates who do have myxedema. I quote:

> "If the normal blood cholesterol be approximately two hundred milligrams, a hypercholesterolemia will be found in true myxedema averaging in a small series of twenty-three cases three hundred milligrams and ranging as high as five hundred milligrams. When these patients are placed upon thyroid extract and the basal metabolism comes to normal, hand in hand with their drop in basal rate goes the drop in blood cholesterol to normal. In those patients with minus degrees of basal metabolism but without clinical evidence of myxedema, there will be a normal or low blood cholesterol when the basal rates are not due to diminished thyroid function and in such cases the feeding of thyroid extract will do no good. The blood cholesterol serves to distinguish the group which has myxedema from the group which does not have myxedema."

From a practical underwriting standpoint, applicants having a persistent low basal metabolic level of minus twenty-four or lower or who show a definite increase in blood cholesterol should be considered unacceptable until under adequate thyroid treatment, and then on a permanent rated basis. Those with a lesser degree of low basal rates should be classified according to their response to thyroid extract and the presence or absence of symptoms. Many, if not most, of the latter group are undoubtedly safely insurable at standard rates.

Doctor Means, in his book on the thyroid, says—"Curative treatment of adult myxedema is as perfect a form of therapy as any known to medicine. So long as the patient takes daily the right amount of thyroid gland or its equivalent in some special preparation, the patient can remain normal and legitimately claim his natural life expectancy." The important point is that he must take thyroid extract for the rest of his life. If he stop, he will in time again become myxedematous. Here, then, is the prime example of the effect of long continued use of thyroid in promoting longevity. Because of the tendency for myxedematous cases to forget the ladder by which they climbed to health, they should in life insurance be considered as permanent substandard risks. We should feel indebted to Doctor Means for his most helpful opinion that thyroid extract, after all, is not a very dangerous drug and that small doses given over a long period of time to non-myxedematous patients would not shorten their lives. This opinion should go far to strengthen our collective underwriting spines. However, there may be many who will hold that a patient's receiving as much as two grains a day constitutes a decided additional risk.

One is impressed with the accumulating evidence that prolonged hyperthyroidism produces no real heart damage provided the heart has not been previously damaged. Furthur, that, when a superimposed hyperthyroidism does produce heart failure and irregularity, it is prima facie evidence that the heart was previously damaged. Lastly, that a patient,

relieved of his thyroid toxicity, has a heart just as good as it was originally. The important underwriting consideration in these cases is to determine what the true heart picture was prior to onset of the hyperthyroidism. Among the many important points to be remembered in developing case histories of hyperthyroidism the following might be stressed: the age of onset; the period of time which elapsed before the condition was recognized and treatment commenced; the presence or absence of associated physical impairments, especially those prior to onset; the response to treatment; and the subsequent history to date. The signs and symptoms of active or recurrent hyperthyroidism to be noted in the examination are too well known for comment here, with the possible exception of emphasizing the importance of an increase in the pulse rate. Frequently, this is the only clue and should have the most careful consideration.

It might be well to mention briefly, in part at least, the role which iodine plays in the treatment of thyroid disorders, with special reference to the epoch-making new work now being done by Doctor Lawrence and his co-workers at the University of California. It has been definitely established that the action of iodine is upon the thyroid gland and not upon the circulating hormone. In simple goiter, the iodine response is upon the local condition of the gland, while in toxic goiter it is not only upon the gland itself, but especially upon the entire organism. In the latter, there is an alteration in metabolism and allied functions, beginning within a few hours, and an equally striking and specific improvement in all symptoms. Advantage is taken of this striking and specific response of toxic goiter to iodine for the purpose of diagnosis and treatment of the condition. By means of the cyclotron, Doctor Lawrence has been able to make iodine radio-active. If this radio-active iodine is given by mouth, it quickly becomes concentrated almost entirely in the thyroid. When it is present in the proper concentration in the thyroid, it causes degeneration of the glandular structure of the thyroid.

This makes possible a new method of treatment of hyperthyroidism, the practical clinical use of which, however, has not yet been worked out.

Time does not permit further discussion of this interesting subject. The future holds much for life insurance in the further development of this field. However, until our knowledge has been placed upon a more solid basis, it is well that we proceed with due caution; in other words, that we stop, look, and listen, that later we may not be ashamed and possibly chagrined over our present enthusiasm.

PRESIDENT FROST—Dr. Means, would you like to make some remarks in closing?

DR. MEANS—I think that everybody's blood sugar is probably fairly low.

I do want to thank the doctor for his discussion. I agree, I think, with all of it.

I was particularly interested in what he said about obesity. I entirely agree with him that most of these fat children, or at least many of them, will straighten out without treatment at all.

Dr. Moore whispered in my ear that some of them are psychopaths, psychotic. That is interesting. That may be important.

I won't take any more of your time. I want to thank you for your patience in listening to my talk.

PRESIDENT FROST—Dr. Means, we are certainly indebted to you for your presentation of this subject.

I want to say to the members of the Association that his coming here represents a definite concession on his part. I didn't know whether I proved to be a good salesman or not; in any event, that would be surprising in a medical director.

When I outlined the problem which I was trying to solve this year, the nature of the possible innovation of the program, in spite of the fact that due to the pressure of his duties he has cut down his speaking materially, he most cordially offered to help me out in putting the program over.

I am also grateful to Dr. Shewbrooks, and I want to thank him for his splendid discussion.

We have now come to the end of the session. It is time for me to yield the reins. I can't do it without making some recognition of those who have helped me in this program. Holding this meeting in Boston had its peculiar problem. Ed Dewis and his most efficient secretary, as usual, have been my mainstay.

Our lovable treasurer, Albert Jimenis, gave me a free hand, and I hope I have not overstepped.

Chester Whitney has done everything possible to help out at this end.

I want to express my appreciation, particularly, also, to the medical directors, my associates, in the Boston companies; every one of them has cheerfully co-operated with me in the effort to make this worthwhile for you. I wish to mention particularly the Committee, Dr. Crawford of the Columbian National Life, Dr. William Davis of the John Hancock and Dr. Davison of the Boston Mutual, and Fred Brown, my associate. Fred Brown, as I said last night, has been my right hand. He is a man to whom you can outline a problem, and then you can forget it, because it will be done.

If you men go back from Boston feeling that you know each other a little better, that you have learned to appreciate some of the lovable qualities of medical directors whom you never met before, and you have come to know them now, if as a body our friendship and understanding has been increased, I shall be well content.

My final duty is to present to you your new President, Dr. Donald Cragin of the Aetna. Dr. Cragin needs no introduction to you. I certainly wish him the best of co-operation and luck during the next year, and I want to give him my pledge of personal support. I know you will give yours in helping him in his activities as President of the Association, and also in the matter of his program next fall.

It becomes my pleasant duty to hand the gavel to Dr. Cragin.

PRESIDENT-ELECT CRAGIN—Dr. Frost, and Members of the Association. I have a few little duties to perform in closing up, but I just want to say that the honor which you have conferred upon the Aetna and myself is the greatest in the medical insurance world. If we can live up to its qualifications, we can be very happy, I know.

I can only say for my company, and also personally, that I thank you with all my heart, and I hope with your help we will have a profitable year together.

Is there any unfinished business?

DR. ROWLEY—I realize the hour is late and no one wants to be detained unnecessarily, but I want to take a moment to present to you a resolution which I trust you may approve.

We all realize our good fortune in having the opportunity of coming to Boston for this meeting. Our social entertainment has been of a very high order, and our scientific entertainment and program have been most interesting and educational.

Without taking more of your time, I want to read to you this resolution, and I will ask the President to call for a vote on it.

"Be it resolved: That the members of this Association present record our appreciation of the hospitality shown us by the New England Mutual Life Insurance Company through its officers; and especially do we wish to record our commendation of our retiring President, Dr. Frost, for planning and presenting to us a program that has been unique, stimulating and broadening in its effect upon our medical underwriting horizon."

PRESIDENT-ELECT CRAGIN—You have heard Dr. Rowley's resolution. I suggest that we approve it unanimously by a rising vote.

(The resolution was passed with a rising vote.)

PRESIDENT-ELECT CRAGIN—In closing, and before asking for the vote for dismissal, I want to thank Dr. Frost for his introduction, and I want to tell him frankly, that he has given me a mark to shoot at next year.

I wish you all a happy, pleasant and safe journey home.

If there is no further business to come before this meeting, a motion is in order to adjourn.

A MEMBER—I move that we adjourn.

* * * * * *

This motion was duly seconded by several of the members present and was carried.

MEMORIALS

DR. HENRY ANTHONY BAKER

1870–1940

Dr. Henry Anthony Baker, Medical Director of the Kansas City Life Insurance Company, died September 17. About six weeks prior to that time he had been stricken with a heart attack but was improving until another attack occurred early in the morning of September 17, which resulted in almost instant death.

Dr. Baker served faithfully and with untiring enthusiasm as medical director of the Kansas City Life for twenty-two years, and due to his discrimination in the selection of risks the Company established a notably low mortality record.

His contact with the fieldmen and Home Office workers was a happy one, always considerate and interested in their problems. He directed in an unassuming spirit of fellowship and, although widely known for his efficiency in insurance matters, the agent or interviewer often carried away the impression that he too had had a voice in the final decision, so simply was it given.

Dr. Baker was born January 2, 1870, in Rochester, New York, and was reared in the home of his great aunt, Susan B. Anthony. His early education was received in Rochester, he later graduated from the College of the City of New York, and afterwards received his Medical Degree from the University of California. Following graduation he accepted an appointment as ship surgeon in the Pacific mail service, during which time he made sixteen trips to the Orient. Afterwards, he followed the private practice of medicine in New York City and later joined the Washington Life Insurance Company as medical director. In 1906 when the Pittsburgh Life and Trust Company took over the business of the Washington Life he was appointed Associate Medical Director.

In 1912 he was made Medical Director of the Pittsburgh Life, which position he held until 1918 when he accepted the office of Medical Director of the Kansas City Life Insurance Company.

He was actively interested in the Medical Section of the American Life Convention, having been present and appointed chairman of the program committee when this body was organized at a meeting of the Convention in Des Moines, Iowa, in 1910. From that time on until his death he served almost continuously; acting as Chairman in 1913, writing papers on various subjects, discussing those presented by other members, conducting the Question Box and giving stereoptican views.

He was a member of the county, state and national medical associations, and an elder in the Presbyterian Church.

DR. JOHN THOMAS JOHNSON BATTLE

1859–1940

Dr. John Thomas Johnson Battle died after a short illness in the early morning of September 30th of coronary occlusion.

He was born in Wake County and spent his early years in and around Wake Forest College, where he received his M. A. degree; then entered the College of Physicians and Surgeons, now combined with the University of Maryland, where he received his M. D. degree in 1884. He practiced awhile at Wadesboro, N. C., where he married Miss Dora Burns in 1896, moving to Greensboro in 1898 to practice, where he soon acquired the confidence of the community and his confreres.

During the World War he was Chairman of the District Advisory Board for military service. He was a member of the Guilford County Board of Health from its organization in 1911.

He was for years a Trustee of both Wake Forest College and Meredith College, and was very active on the Board of the latter at the time of his death.

He was one of the pioneers of life insurance in Greensboro.

He was Medical Director of the Greensboro Life Insurance Company 1905–1912, when he became Medical Director of the Southern Life and Trust Company (Pilot Life Insurance Company). He served in that capacity until 1918 when he joined the Jefferson Standard Life Insurance Company as Medical Director.

He was always a constant attendant at the meetings of his County and State Medical Societies, and for years has been an Honorary Member of both Societies; served on the Board of Medical Examiners of the State from 1902 to 1908.

He was a perfect example of a Christian gentleman and like St. Luke a beloved physician.

DR. HENRY WIREMAN COOK

1877–1940

"In the midst of Life, we are in Death."

How vividly and sorrowfully this prophecy was realized by the members of the family, the associates in his Company, and the numerous friends and admirers of Henry Wireman Cook, M. D., on April 25, 1940.

On that morning he arrived at his office, apparently in good health, and with his usual cheerful greetings to all. At 10:45 o'clock he had an attack of coronary occlusion, and one hour later his earthly life flame had been snuffed out.

Those who were privileged to know and come in close contact with him, and especially those who could be numbered among his friends, know what an active and full life he lived, that he was courageous and upright in all his dealings with his fellow man, and that he established an undying example of humaneness, industriousness, and ability that many strive for but few attain.

Possessor of an extraordinary retentive and logical mind, and being an untiring student and investigator, he readily rose to the top in all of his undertakings and received many honors.

Born in Baltimore on November 8, 1877, Doctor Cook re-

ceived the A. B. degree from the Johns Hopkins University in 1898 and his M. D. from its Medical School in 1902. He practiced in Richmond, Virginia, was appointed Medical Referee for the Mutual Life Insurance Company of New York in 1903, and while in Richmond attained his greatest honor by winning one of her fairest daughters as his wife. In 1906 he was appointed Medical Director of the Northwestern National Life Insurance Company and elected Vice President in 1913.

He was one of the first to realize the importance of blood pressure observations in the evaluation of cardiovascular conditions and assisted in developing and popularizing the Riva Rocci apparatus.

He was a member and outstanding leader in the Medical Section of the American Life Convention, and its interests and progress were very dear to him. From 1922 to 1927, and again in 1936 he was Chairman of its Disability Committee, and Chairman of the Medical Section for the year 1923–24.

He was President of the Life Office Management Association in 1926 and 1927 and Vice President of the American Management Association in 1928.

During the World War he was in Washington, D. C., as Personnel Director of the American Red Cross.

He was active in Civic and Public Health affairs as a member of the Board of Directors of the Minneapolis Council of Social Agencies and of its Community Health Service.

In 1914 he became a member of the Association of Life Insurance Medical Directors, and his extensive and accurate knowledge with regard to clinical and insurance medicine, and the correlation of the one with the other, caused him to be called upon frequently for a paper, or a discussion or to serve on some important Committee. Up to and including the year 1936, he presented papers or studies sixteen times, and was only exceeded in this respect by one other member, Doctor O. H. Rogers. His discussion in Volume XXV (1938) on Rheumatic Heart Disease, and his Presidential Address in Volume XXVI (1939) are masterpieces and show his accurate knowledge and clear and succinct presentation of the subject,

and his broad and tolerant vision regarding the proper relationship between the Agency force, the Examiner in the field, and the Selection Department at the Home Office.

He was elected President of this Association in 1939 and represented our organization at the Meeting of the International Life Insurance Medical Congress in Paris last May. He was a frequent speaker at Life Insurance gatherings throughout the Country, and was universally known and admired.

He, in collaboration with his son Henry, has recently written and had published a book on the Grouping of Medical Impairments and Factors regarding the Importance of Co-operation between the Agency Force and the Selection Department. This is an excellent compilation of his views and experience.

Yes, and it is worthy of repetition, Henry Wireman Cook, led a serviceable and an abundant life.

He was a highly valued and respected member of this Association.

He will be missed not only as a scholar and contributor, but we will bear in mind the constant presence, in mien, dress, and dignity, of a gentleman of the "Old School" who held "Honesty of Conscience above Honesty of Purse", and without ostentation or complaint pursued his daily tasks.

DR. DONALD W. SKEEL

1873–1940

Dr. Donald W. Skeel, Medical Director of Occidental Life Insurance Company, was born September 26, 1873 at Granville, Illinois, the son of Nathan L. and Mary Skeel, where he spent his childhood and received his early schooling. While still a lad he moved to California and ultimately entered the Medical College of the University of Southern California at Los Angeles. This institution conferred its degree of Doctor of Medicine upon him in 1906. He earned his way through

college by long and arduous work as a nurse, and always felt that this experience, hard as it was, formed one of the most valuable backgrounds for his later professional activities. As a result of this service Dr. Skeel was made an interne at Los Angeles County Hospital during the last year of his medical course. It is rare indeed for a man to be so recognized a year before his graduation and the fact testifies to the quality of his work and the regard in which he was held by the medical staff of the hospital. During his earlier professional years Dr. Skeel enjoyed a large and lucrative practice, much of it among the pioneer families of Los Angeles. Many of the patients of those years remained his warm friends as long as they lived.

Occidental Life Insurance Company was founded in June, 1906 and one of the first medical examiners appointed was young Doctor Skeel. He continued to be identified with this Company until his death and his devotion and industry in its behalf have played an important part in the growth and progress Occidental has made in the intervening thirty-four years. He saw it develop from an insignificant beginning to an institution with more than seventy millions of assets and over five hundred millions of insurance in force. None of its officers was prouder of this record, nor with more justification when the Company's consistently low mortality rate is considered.

On January 1, 1923, Dr. Skeel was made Assistant Medical Director and in October 1927 he was advanced to the post of Medical Director. A large portion of the company's business in force has had his personal sharp scrutiny, whether as examiner, referee or medical director. He was long a member of the Company's operating, risks and claims committees.

Dr. Skeel was a charter member of the Medical Symposium Society, Los Angeles, and the Los Angeles Obstetrical Association, as well as an active member of many other professional groups, including the American Medical Association, the California Medical Association, the Los Angeles County Medical Society, the Medical Directors Association of

Life Insurance Companies and the Medical Section of the American Life Convention. He was also a member of Nu Chapter of Nu Sigma Nu and of the University Club of Los Angeles. Also, when his country needed physicians and surgeons in the World War, Dr. Skeel was prompt to respond and served as a captain in the Medical Corps throughout that conflict.

A busy man, a quiet but effective worker, Dr. Skeel added to these essential qualities a rare personality that was reflected in his every act and word. In an organization that at the time of his death numbered over six hundred officers and employees, exclusive of agents, Dr. Skeel accomplished the seemingly impossible feat of establishing a personal relationship with almost every fellow worker. He watched over their health and advised with them in their personal troubles in the same unobstrusive way in which he pursued his official activities.

He loved his work and the host of friends that work created. After a short illness, which at first gave little evidence of its seriousness, he suddenly passed away at his home in Los Angeles on October 11, 1940. He was at his office the day before his death and so almost literally died "in harness", probably the end he would have chosen. He is survived by his wife, Mrs. Mabel Skeel and his sister, Miss Florence Skeel.

The Occidental Life Insurance Company grew and flourished greatly under Dr. Skeel's wise direction of its medical underwriting. Even more enduring is the privilege of friendship with him that is treasured by hosts of fellow workers, patients and the many circles of men and women whom he enriched with his presence.

DR. GEORGE ALEXANDER VAN WAGENEN

1845–1940

Dr. George A. Van Wagenen, for nearly half a century a Medical Director of the Mutual Benefit Life Insurance Company

of Newark, New Jersey, died on January 2, 1940, at his winter home in St. Petersburg, Florida. He was ninety-four years of age at the time of his death.

Dr. Van Wagenen was of Dutch descent and a native of Newark, New Jersey, in which city he resided throughout his lifetime until the infirmaties of advanced age caused him to seek an abode in the Southland. During his boyhood days his parents moved from their home on the Passaic River to Roseville, which was then a small village quite detached from Newark. He was exceedingly fond of his native city. He received his early education in the Newark Academy and in 1868 he was graduated from Princeton College—now Princeton University. At the time of his death he was one of the oldest of the Princeton alumni. Following his collegiate studies he entered the College of Physicians and Surgeons in New York City. After his graduation he engaged in the practice of medicine in Newark and was an active member of the staff of St. Michael's Hospital.

Dr. Van Wagenen's scientific mind, his genial personality and his sympathetic attitude made him an ideal family physician. Few physicians in Newark have been held in higher esteem or in more affectionate memory.

In 1878 Dr. Van Wagenen became a member of the Medical Board of the Mutual Benefit and in 1896 he was appointed Medical Director in which capacity he served until a few years prior to his death. On May 29, 1889, Dr. Van Wagenen, in conjunction with twenty-seven other physicians, organized The Association of Life Insurance Medical Directors and at the time of his death he was the sole surviving charter member of this Association. In this organization, as in all others to which he belonged, he was very highly esteemed by his associates.

But those of us who knew him best will ever remember Dr. Van Wagenen not only as a physician who honored his high calling, but also and more particularly as a genial associate and loyal friend. It was a real privilege to enjoy his companionship day by day, to listen to his words of wisdom and

to his homely precepts. He had the opportunity of extensive travel and was very fond of music, drama and the fine arts. His philosophy of life can best be appreciated by a reference to some of his own words. In a letter written several years prior to his death he wrote:

> "The crown of old age may be beautiful, but it is very heavy. Do not think that I am giving up the ship for even though weak in body, my old head is in fair shape for eighty-eight."

In another letter written some years later he said:

> "One should be thankful he can read, have a good memory, eat, sleep and enjoy the sunshine, with plenty of kind friends and enough to supply his creature wants without worry."

He has bid us adieu but he will ever live in the esteem and affection of all those who knew him.

DR. FANEUIL SUYDAM WEISSE

1875–1940

It is with deepest regret that we bring to your attention the death, on February 24, 1940, of Doctor Faneuil Suydam Weisse, former Medical Director of The Mutual Life Insurance Company of New York. He died at Winter Park, Florida, after a long illness.

Doctor Weisse was born in New York City on April 20, 1875, and received his preliminary education at the Berkeley School in that city. He was graduated from Columbia College with the degree of A. B. in 1897, and received his Medical Degree from the College of Physicians and Surgeons, Columbia University in 1900. Doctor Weisse served his internship at Roosevelt Hospital and after various clinical appointments became, in 1902, Assistant Attending Surgeon at the State Hospital for Crippled Children at Tarrytown, New York.

Doctor Weisse came to The Mutual Life Insurance Company in 1904 as a Home Office Medical Examiner. He was appointed Medical Director in 1911 and in 1925 became Senior

Medical Director. He served in that position until February, 1938, when he retired because of ill health. In 1906, Doctor Weisse was elected to membership in the Association of Life Insurance Medical Directors and held the office of Secretary from 1912 to 1918, Vice-President 1918–1919 and President 1919–1920. He served as a member of various Committees in the Association, including the M. I. B. Committee. During the time he was a member of this Association, he contributed various papers and discussions at the meetings. Doctor Weisse also acted in a consulting capacity for a number of endowment institutions, particularly in connection with the Episcopal Church.

Doctor Weisse had two pleasant hobbies, reading and fishing, to which he devoted his leisure time. In addition to being a member of the Association of Life Insurance Medical Directors, Doctor Weisse was also a member of the American Medical Association, the New York County and New York State Medical Societies, the Anglers Club and the University Club of New York. He is survived by his wife, Margaret Mason Young Weisse, whom he married on June 1, 1904, and by a brother, H. Bethune Weisse, of White Plains, New York.

DECEASED MEMBERS

John L. Adams, M. D.	New York, N. Y.
Charles D. Alton, M. D.	Hartford, Conn.
Malcolm O. Austin, M. D.	San Francisco, Calif.
Walter C. Bailey, M. D.	Boston, Mass.
Henry A. Baker, M. D.	Kansas City, Mo.
A. W. Barrows, M. D.	Hartford, Conn.
John T. J. Battle, M. D.	Greensboro, N. C.
Wesley W. Beckett, M. D.	Los Angeles, Calif.
Charles D. Bennett, M. D.	Newark, N. J.
Charles Bernacki, M. D.	New York, N. Y.
Thomas W. Bickerton, M. D.	New York, N. Y.
Albert W. Billing, M. D.	New York, N. Y.
Robert J. Blanchard, M. D.	Winnipeg, Man.
Frederick G. Brathwaite, M. D.	New York City
William R. Bross, M. D.	New York, N. Y.
Chauncey R. Burr, M. D.	New York, N. Y.
Robert L. Burrage, M. D.	Newark, N. J.
James Campbell, M. D.	Hartford, Conn.
Willard B. Carpenter, M. D.	Columbus, Ohio
Frank W. Chapin, M. D.	New York City
Frederick W. Chapin, M. D.	Springfield, Mass.
Ferdinand E. Chatard, M. D.	Baltimore, Md.
Henry Colt, M. D.	Pittsfield, Mass.
Henry W. Cook, M. D.	Minneapolis, Minn.
Thomas C. Craig, M. D.	New York, N. Y.
Edward Curtis, M. D.	New York, N. Y.
Clark W. Davis, M. D.	Cincinnati, Ohio
William B. Davis, M. D.	Cincinnati, Ohio
Charles A. Devendorf, M. D.	Detroit, Mich.
Henry K. Dillard, M. D.	Philadelphia, Pa.
Frank Donaldson, M. D.	Baltimore, Md.
Percy G. Drake, M. D.	Hartford, Conn.
Edwin W. Dwight, M. D.	Boston, Mass.
James B. Eagleson, M. D.	Seattle, Wash.
Z. Taylor Emery, M. D.	New York, N. Y.
John W. Fisher, M. D.	Milwaukee, Wis.

DECEASED MEMBERS

Paul FitzGerald, M. D.	Newark, N. J.
Thomas A. Foster, M. D.	Portland, Me.
Samuel W. Gadd, M. D.	Philadelphia, Pa.
Homer Gage, M. D.	Worcester, Mass.
Thomas H. Gage, M. D.	Worcester, Mass.
Donald M. Gedge, M. D.	New York, N. Y.
Walter R. Gillette, M. D.	New York, N. Y.
Frank S. Grant, M. D.	New York, N. Y.
Frederick L. Grasett, M. D.	Toronto, Can.
Landon Carter Gray, M. D.	New York, N. Y.
Frederick W. Hagney, M. D.	Newark, N. J.
Ignatius Haines, M. D.	Boston, Mass.
George C. Hall, M. D.	Richmond, Va.
Joseph B. Hall, M. D.	Hartford, Conn.
Edward H. Hamill, M. D.	Newark, N. J.
William W. Hitchcock, M. D.	Los Angeles, Calif.
Angier B. Hobbs, M. D.	New York City
Donald C. Hoffman, M. D.	New York City
Edgar Holden, M. D.	Newark, N. J.
John Homans, M. D.	Boston, Mass.
John Homans, 2d, M. D.	Boston, Mass.
J. Charles Humphreys, M. D.	Philadelphia, Pa.
Abel Huntington, M. D.	New York, N. Y.
Ross Huston, M. D.	Des Moines, Iowa
Henry H. Hutchison, M. D.	Toronto, Can.
Lefferts Hutton, M. D.	New York City
Phineas H. Ingalls, M. D.	Hartford, Conn.
Arthur Jukes Johnson, M. D.	Toronto, Ont.
John M. Keating, M. D.	Philadelphia, Pa.
Edward B. Kellogg, M. D.	Boston, Mass.
William W. Knight, M. D.	Hartford, Conn.
Edward Lambert, M. D.	New York, N. Y.
John B. Lewis, M. D.	Hartford, Conn.
Ernest H. Lines, M. D.	New York, N. Y.
John M. Little, M. D.	Boston, Mass.
Robert L. Lounsberry, M. D.	Binghamton, N. Y.

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Dominion Life Assurance Co., Waterloo, Canada.	A. J. McGanity, M. D.

Equitable Life Assur. Soc., New York City.	E. W. Beckwith, M. D. Leslie Brown, M. D. R. M. Daley, M. D. P. G. Denker, M. D. O. W. King, M. D. F. W. McSorley, M. D. H. L. Mann, M. D. E. W. Scott, M. D. A. L. Sherrill, M. D. W. A. Smith, M. D. N. A. Sullo, M. D. B. C. Syverson, M. D. H. E. Ungerleider, M. D.
Equitable Life Insurance Co., Washington, D. C.	J. L. Brooks, M. D.
Equitable Life Insurance Co. of Canada, Waterloo, Can.	W. L. Hilliard, M. D.
Equitable Life Insurance Co. of Iowa, Des Moines, Iowa.	W. O. Purdy, M. D. R. R. Simmons, M. D.
Excelsior Life Ins. Co., Toronto, Canada.	W. E. Ferguson, M. D.
Federal Life Insurance Co., Chicago, Ill.	F. L. B. Jenney, M. D.
Fidelity Mutual Life Insurance Co., Philadelphia, Pa.	J. T. Sheridan, M. D. J. L. Siner, M. D. I. G. Towson, M. D.
Franklin Life Insurance Co., Springfield, Ill.	Frederick Fink, M. D.
General American Life Insurance Co., St. Louis. Mo.	J. H. Ready, M. D.
Great Southern Life Insurance Co., Houston, Texas.	J. E. Daniel, M. D.
Great West Life Assurance Co., Winnipeg, Canada.	W. L. Mann, M. D.
Guarantee Mutual Life Co., Omaha, Neb.	J. P. Donelan, M. D.

Guardian Life Insurance Co., New York City.	M. B. Bender, M. D. B. C. Bullen, M. D. D. C. Roberts, M. D.
Gulf Life Insurance Co., Jacksonville, Fla.	B. H. Malone, M. D.
Home Life Insurance Co., New York City	J. H. Humphries, M. D. G. E. Woodford, M. D.
Home Life Insurance Co. of America, Philadelphia, Pa.	H. W. Goos, M. D.
Imperial Life Assurance Co., Toronto, Canada.	H. B. Anderson, M. D. R. W. Mann, M. D.
Jefferson Standard Life Ins. Co., Greensboro, N. C.	
John Hancock Mutual Life Ins. Co., Boston, Mass.	W. B. Bartlett, M. D. R. A. Behrman, M. D. W. L. Davis, M. D. Byam Hollings, M. D. Robert Sanderson, M. D.
Kansas City Life Insurance Co., Kansas City, Mo.	J. E. Bee, M. D.
Life & Casualty Insurance Co., Nashville, Tenn.	C. T. Kirchmaier, M. D.
Life Insurance Co. of Virginia, Richmond, Va.	E. S. Williams, M. D.
Lincoln National Life Insurance Co., Fort Wayne, Ind.	H. C. McAlister, M. D. W. E. Thornton, M. D.
London Life Ins. Co., London, Canada.	J. T. Bowman, M. D. A. S. Graham, Jr., M. D.
Manhattan Life Insurance Co., New York City.	G. H. Barber, M. D.
Manufacturers Life Insurance Co., Toronto, Canada.	H. C. Cruikshank, M. D. R. C. Montgomery, M. D.
Maryland Life Insurance Co., Baltimore, Md.	G. C. Lockard, M. D.

Massachusetts Mutual Life Ins. Co., Springfield, Mass.	R. B. Andrews, M. D. H. B. Brown, M. D. L. D. Chapin, M. D. R. B. Ober, M. D. Gordon Ross, M. D. Morton Snow, M. D.
Metropolitan Life Insurance Co., New York City.	C. C. Berwick, M. D. N. R. Blatherwick, M. D. E. C. Bonnett, M. D. A. W. Bromer, M. D. C. L. Christiernin, M. D. J. A. Evans, M. D. R. K. Farnham, M. D. H. H. Fellows, M. D. J. C. Horan, M. D. A. O. Jimenis, M. D. H. B. Kidd, M. D. R. J. Kissock, M. D. A. J. Lanza, M. D. S. W. Means, M. D. W. P. Reed, M. D. G. P. Robb, M. D. Wallace Troup, M. D. H. B. Turner, M. D. E. W. Wilson, M. D.
Midland Mutual Life Ins. Co., Columbus, Ohio	A. R. Stone, M. D.
Minnesota Mutual Life Ins. Co., St. Paul, Minn.	T. H. Dickson, M. D.
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National Life Assurance Co., Toronto, Canada.	E. C. Noble, M. D.
National Life Insurance Co., Montpelier, Vt.	G. E. Allen, M. D. E. A. Colton, M. D. A. J. Oberlander, M. D.
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New York Life Insurance Co., New York City.	H. E. Bogart, M. D. William Bolt, M. D. E. J. Campbell, M. D. F. C. Evers, M. D. R. A. Fraser, M. D. E. M. Freeland, M. D. E. E. Getman, M. D. W. D. Heaton, M. D. I. C. Lawler, M. D. R. W. Pratt, M. D. W. C. Roberts, M. D. O. H. Rogers, M. D. R. L. Weaver, M. D. G. R. Welch, M. D.
North American Life Assur. Co., Toronto, Canada.	T. D. Archibald, M. D. J. G. Falconer, M. D.

North American Life Ins. Co., Chicago, Ill.	C. B. Irwin, M. D.
Northern Life Ins. Co., Seattle, Wash.	
Northwestern Mutual Life Ins. Co., Milwaukee, Wis.	R. W. Benton, M. D. R. H. Feldt, M. D. R. T. Gilchrist, M. D. W. G. Hyde, M. D. D. F. Rikkers, M. D. A. W. Sivyer, M. D. G. F. Tegtmeyer, M. D. D. E. W. Wenstrand, M. D.
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Provident Mutual Life Ins. Co., Philadelphia, Pa.	E. J. Dewees, M. D. Herbert Old, M. D.
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Security Mutual Life Ins. Co., Binghamton, N. Y.	W. B. Aten, M. D.
Shenandoah Life Insurance Co., Roanoke, Va.	D. S. Garner, M. D.

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